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Full Name of Author — Nom complet de l'auteur

Meredith Anne Galbraith

Date of Birth — Date de naissance

October 25, 1958

Country of Birth — Lieu de naissance

Canada

Permanent Address — Résidence fixe

1108 Johnson St., Kingston, Ont. K7M 2N6

Title of Thesis — Titre de la thèse

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Name of Supervisor — Nom du directeur de thèse

Angelo N. Belcastro

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EXERCISE AND REPRODUCTIVE FUNCTION: THE EFFECTS OF STRENGTH TRAINING ON SERUM GONADOTROPIN LEVELS

by

© MEREDITH ANNE GALBRAITH

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

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PERMANENT ADDRESS:

1108 Johnson St

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DATED July 21 1983

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled EXERCISE AND REPRODUCTIVE FUNCTION: THE EFFECTS OF STRENGTH TRAINING ON SERUM GONADOTROPIN LEVELS submitted by MEREDITH ANNE GALBRAITH in partial fulfilment of the requirements for the degree of MASTER OF SCIENCE.

A. N. Belcastro

Supervisor

W. C. Sykes

Date July 21, 1983

Abstract

To determine the gonadotropin response to resistance exercise and to observe the effect(s) of resistance training on the response, frequent blood samples were obtained during a Nautilus exercise session in seven women before and after eight weeks of training. Blood samples were obtained through an indwelling intravenous catheter for 1/2 hour before exercise, one hour of exercise, and 1/2 hour of recovery from exercise. Exercise consisted of three sets of 8 - 12 repetitions on each of three upper body and three lower body exercises. Blood samples were analyzed for hematocrit, lactate, LH, and FSH. Both LH and FSH increased with resistance exercise in the pre- and post- tests, but only LH increased significantly (47% above baseline in the pre-test and 60% above baseline in the post-test). A biphasic LH response was observed in both the pre-test and the post-test with one peak occurring during upper body exercise and a second peak occurring during lower body exercise. The LH response after training was delayed compared to the pre-test but remained higher over the exercise session. The results of the study suggest that the LH response i) was not a consequence of hemoconcentration (hematocrit increased < 10%), ii) did not appear to be related to either muscle mass or the amount of work performed, iii) may be related to the relative intensity of the exercise, iv) appears to be related to the sequence of the two groups of exercises, and v) may be altered by training.

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I. INTRODUCTION

The high incidence of oligomenorrhea and amenorrhea among female athletes suggests a causal relationship between regular strenuous physical activity and menstrual dysfunction. While the physiological consequences of training which are conducive to amenorrhea are unknown, youth, nulliparity, and prior menstrual irregularity appear to influence susceptibility to the condition (Speroff and Redwine, 1980; Baker et al., 1981; Schwartz et al., 1981; Shangold and Levine, 1982). Decreased and/or low body fat (Frisch et al., 1980; Schwartz et al., 1981), physical stress (Warren, 1980; Anderson, 1979), and psychological stress (Schwartz et al., 1981; Shangold and Levine, 1982) have all been implicated in the etiology.

Exercise-induced aberrations in menstrual function are likely manifestations of altered neurohumoural input at the hypothalamus-pituitary-ovarian axis. However, studies evaluating circulating gonadotropin levels with acute exercise have produced conflicting results. Bonen and associates (1979) found no change in either luteinizing hormone (LH) or follicle-stimulating hormone (FSH) levels over an acute exercise bout on a bicycle ergometer. Jurkowski and associates (1978) reported an increase in FSH levels over an acute exercise bout in the mid-follicular phase of the menstrual cycle but no change in FSH with exercise in the mid-luteal phase and no change in LH in either phase. Cumming and associates (1981) not only found a significant increase in both LH and FSH levels with exercise but also an increase in LH levels in anticipation of exercise. These contradictory results likely reflect the differences in experimental protocols. The time of day, the phase of the menstrual cycle, the exercise regime employed, plus the mode, timing and frequency of blood sampling could all have a bearing on the results.

The data concerning the resting gonadotropin levels of amenorrheic athletes is likewise conflicting. Amenorrheic runners, when compared to normally-cycling runners and non-runners, have demonstrated; i) low to low-normal LH (Dale et al., 1979; Baker et al., 1981) and FSH concentrations (Dale et al., 1979), ii) comparable LH and FSH concentrations (Cumming et al., 1981), and iii) high LH concentrations (Schwartz et al., 1981). Again, the discrepant results may be explained in part by variations in testing methodology.

Thus, it is uncertain how and to what degree exercise and/or training alter LH and FSH levels. Furthermore, it is not clear to what degree alterations in body fat composition may influence the results. Low body fat and/or weight loss have been associated with amenorrhea (Wentz, 1980), exercise (Speroff and Redwine, 1980), and altered concentrations of some reproductive hormones (Fishman et al., 1975; Wentz, 1980). Effects resulting from exercise-induced changes in fat composition have yet to be clearly dissociated from effects resulting from exercise alone.

Strength training programs are generally associated with smaller decreases in fat weight than are endurance training programs (Wilmore, 1983). Strength training programs provide one method of studying the effects of increased physical stress without a significant alteration in body fat. It is the purpose of this study to determine the gonadotropin response to an acute resistance exercise bout and to observe whether the response is altered following a resistance training program.

LIMITATIONS

Limitations of the study were as follows;

1. Human subjects: Subject compliance was an integral factor in the successful completion of the study.

2. Small sample size: Although several women volunteered to be subjects for the study, many did not pass the criteria for selection (see Methods). Fifteen women were chosen as subjects, ten decided to begin the study, but only seven completed it.

3. Lack of a control group: The seven subjects served as their own controls as they were tested twice, once before and once after eight weeks of training. A control group which could be tested twice, once before and once after the same eight week period but which would not undergo the training, would have been desirable.

However, not only would it have been extremely difficult to find a group of women who fitted the selection criteria who would be willing to undergo the pre-test and post-test without the training, but it also would have been extremely difficult to schedule their tests. Testing had to be performed during the early follicular phase of the menstrual cycle and at only one time of day (3.00 p.m.). It was already awkward scheduling the tests of the training subjects.

4. Weight training: Problems inherent in weight training studies include difficulties in evaluating work performance and the subjective assessment of maximal work capacity.

5. Estimation of residual volume: Helium or oxygen dilution techniques are the methods of choice for accuracy in the assessment of residual lung volume. These techniques were not available. However, errors due to inaccurate estimation of residual volume should not have influenced the results since a change in fat content, rather than abolute fat content, was the parameter of interest and since residual volume would not be expected to change with training, it should influence pre-test and post-test results equally.

Table 1: Gonadotropin Results in the Literature

A. Gonadotropin Levels with Exercise

Study	LH	FSH
Jurkowski et al., 1978	no change	increased
Bonen et al., 1979	no change	no change
Cumming et al., 1981	increased	increased

B. Gonadotropin Levels of Amenorrheic Athletes

Study	LH	FSH
Dale et al., 1979	low	low
Baker et al., 1981	low	low
Cumming et al., 1981	normal	normal
Schwartz et al., 1981	high	

II. METHODS

Ten female university students selected from a large volunteer population gave informed consent and served as subjects. Criteria for selection required that the women i) had no known health problems, ii) were not using oral contraceptives, iii) were not on any special diet, iv) had relatively regular menstrual function, and v) were not highly trained or in training. Three subjects dropped out during the course of the study. Physiological and gynecological characteristics of the remaining seven subjects are presented in Table 2.

The experimental design involved a pre-training test, training sessions, and a post-training test. Training sessions were held three times per week for approximately eight weeks. Each subject completed a minimum of twenty-four sessions. Each session consisted of three sets each of three upper body exercises (pullover, armcross, decline press) and three lower body exercises (hip and back, quadriceps, leg curls) on Nautilus equipment. A set involved eight to ten repetitions for upper body exercises or ten to twelve repetitions for lower body exercises. Resistance was increased when a subject had successfully completed all three sets for three consecutive training days (ie. three sets of ten repetitions each for upper body exercises, three sets of twelve repetitions each for lower body exercises).

Subjects were studied over an exercise session during the early follicular phase of the menstrual cycle. The time course of the testing protocol is illustrated in Figure 1. Testing sessions began at 3:00 p.m. with the insertion of an indwelling intravenous catheter with saline drip into a wrist vein. After a one hour normalization period with the subject seated in the weight room, three resting venous blood samples of approximately 10 mls each were taken at 15 minute intervals. At 4:30 p.m. the subject began exercising and blood samples were drawn at the end of the third set of each exercise (i.e. at approximately 10 minute intervals). Two recovery blood samples were taken 15 minutes and 30 minutes after the cessation of exercise.

At the time of sampling hematocrit was determined in unheparinized micro-hematocrit tubes. One ml of blood was added to ice-cold perchloric acid to obtain a protein-free filtrate for lactic acid analysis by the enzymatic method of the Sigma Chemical Company. The remainder of

TABLE 2: PHYSIOLOGICAL AND GYNECOLOGICAL CHARACTERISTICS OF SUBJECTS

Subject	Age (years)	Height (cm)	Weight (Kg)	Menarcheal Age (years)	Gynecological Age (years)	Weight at Menarche (Kg)	Cycle Periodicity (days)	Duration of Menses (days)
A	20	160	56.4	11	9	48	23	5
B	18	173	63.6	14	4	52	27	3
C	24	160	59.1	10	14	46	23	6
E	24	160	47.3	13	11	42	31	6
F	26	160	60.9	14	12	58	28	6
H	19	173	55.9	15	4	52	29	7
I	21	173	63.2	13	8	62	23	5
Mean	22	165	58.1	13	9	51	26	5
SEM	3	7	6	1	4	7	3	1

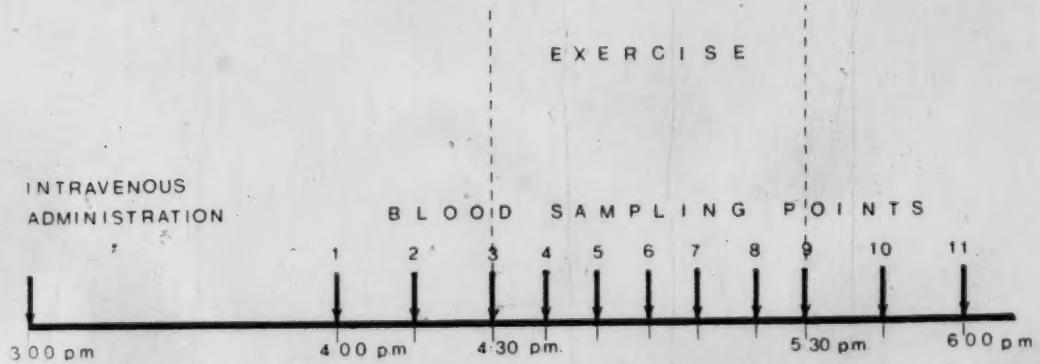

Figure 1 Time course of the testing protocol

the blood was left to clot at room temperature and the serum removed for hormone analysis. Luteinizing hormone and follicle-stimulating hormone were analyzed by Amerlex radioimmunoassay (Amersham Co., Oakville, Ontario).

The assay procedures depend on competition between hormone in sample or standard and labeled hormone for a limited number of binding sites on a hormone-specific antibody (LH-specific antibody or FSH-specific antibody). The proportion of labeled hormone bound to antibody is inversely related to the concentration of unlabeled hormone present in the standard or sample. The antibody-bound hormone was reacted with Amerlex second antibody reagent containing second antibody bound to polymer particles of uniform diameter. Separation of the antibody-bound fraction was effected by centrifugation and removal of the supernatant. The hormone concentration in the samples was determined by measuring the proportion of labeled hormone bound in the presence of reference standard solutions containing known quantities of hormone.

Percent body fat was determined both before and after involvement in the training program using the hydrostatic method and the formula of Brozek and associates (1963). A Collins Vitalometer was used to measure vital capacity. Underwater weighing was completed with the subject holding a maximal inspiration.

A two-way analysis of variance was conducted on all hormone and lactate data. T-tests were performed on the results of the strength and body fat tests. Statistical significance was accepted at $p<0.05$.

III. RESULTS

The weekly progression in the training program is illustrated in Figure 2. The training program was successful ($p<0.01$) as demonstrated by the number of plates lifted during the post-test compared to the pre-test (Figure 3). There was no significant alteration in body composition ($p>0.05$) over the 8 weeks of training (Table 3; Figure 4).

Hemoconcentration occurred with resistance exercise as indicated by hematocrit values. The pattern of change was similar in the pre-test and post-test with peak increases of 9% above resting values being reached during exercise, and a return to baseline occurring within 15 minutes of stopping exercise (Figure 5: Mean + S.E.M.)($p<0.05$).

Blood lactate concentrations showed an immediate increase with exercise, levelling out during the three upper body exercises and then reaching a second higher peak during the three lower body exercises (Figure 6: Mean + S.E.M.). Pre-test and post-test lactate values were not significantly different during rest (4.70 ± 1.01 mg% compared to 3.90 ± 1.16 mg%) or during the three upper body exercises (26.02 ± 8.19 mg% compared to 25.58 ± 8.48 mg%). However, the lactate concentrations obtained over the three lower body exercises were significantly higher during the post-test (peak $=61.26\pm 13.8$ mg%) than the pre-test (peak $=43.36\pm 10.90$) and remained higher over recovery ($p<0.05$)(Figure 6).

There was a significant increase in LH concentrations with acute exercise in both the pre-test and post-test (Figure 7). In both cases the pattern was biphasic involving an initial peak after the second upper body exercise (Time = 20 min.), a decrease (Time = 30 min.), and then a second peak after the second lower body exercise (Time = 50 min.). The patterns were not, however, identical. In the pre-test, LH levels rose immediately, reaching significance after the first upper body exercise, then peaking after the second upper body exercise. In the post-test the rise was delayed, not reaching significance until after the second upper body exercise. Still, there was no significant difference between pre-test and post-test peak values at any of the time points studied (Table 4). The mid-bout decrease in LH levels rose again more gradually to a second, lower peak after the second lower body exercise. In the post-test, LH levels rose again more

rapidly, reaching significance after the first lower body exercise and attaining a second, higher peak after the second lower body exercise. Again there was no significant difference between pre-test and post-test peak values.

The differences in the pre-test and post-test LH responses were more evident when the results were expressed in terms of a percentage of baseline values (Figure 8). While LH levels peaked earlier in the pre-test, the peak values obtained were only 47% and 32% above baseline values compared to 60% in the post-test. In the pre-test the mid-bout decrease brought LH levels almost back to baseline, while in the post-test LH levels remained 50% above baseline values.

FSH did not alter significantly from baseline values with exercise and the response was not influenced by training (Figure 9; Table 5). However, the FSH results expressed in terms of a percentage of baseline values suggest that FSH does increase with exercise in a pattern similar to, although lower than, LH (Figure 10). Moreover, post-test values were consistently higher than pre-test values. Neither of these results proved significant however, this is likely due to the vast intra-individual difference for this hormone.

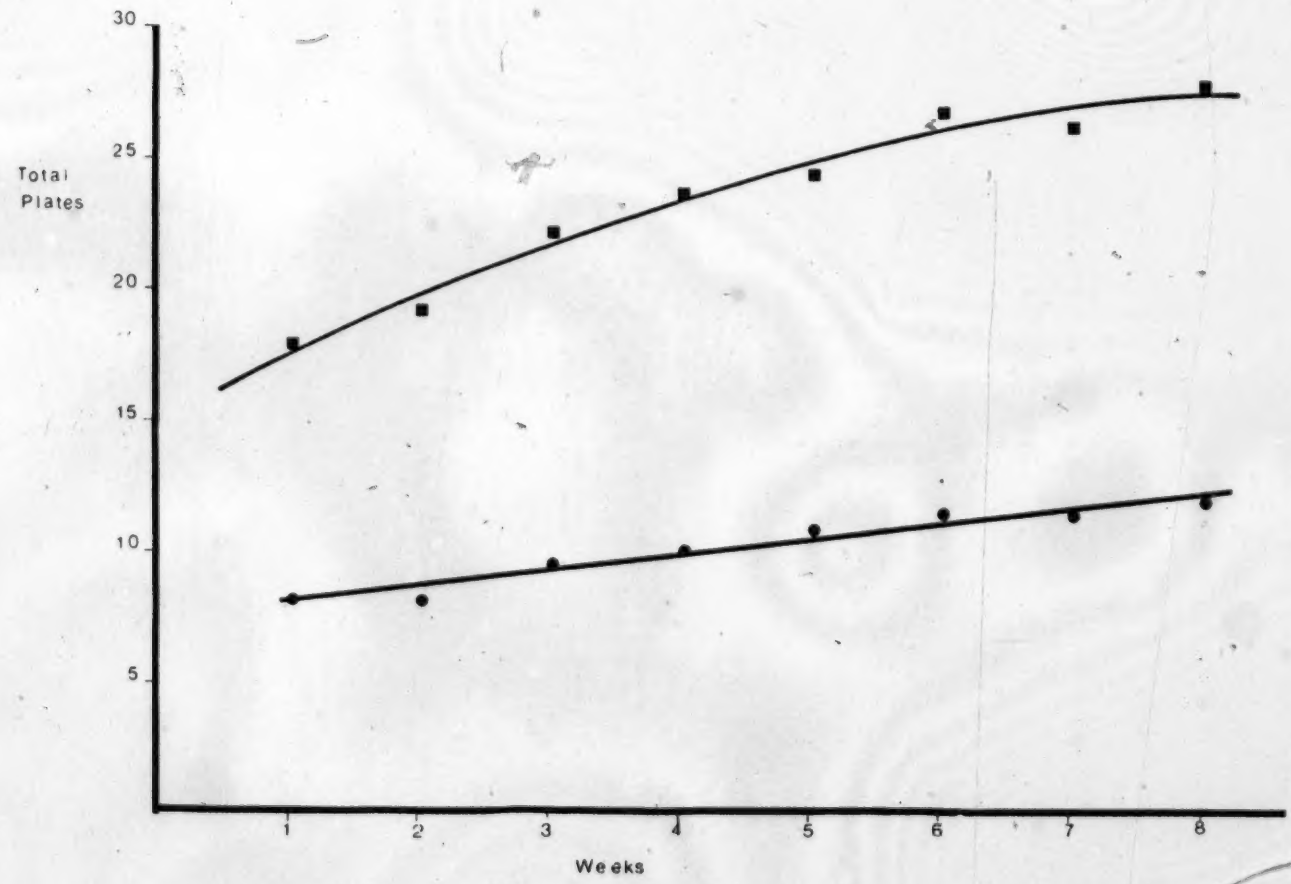

Figure 2 Total plates for the three upper body exercises combined (●) and the three lower body exercises combined (■), showing weekly progression

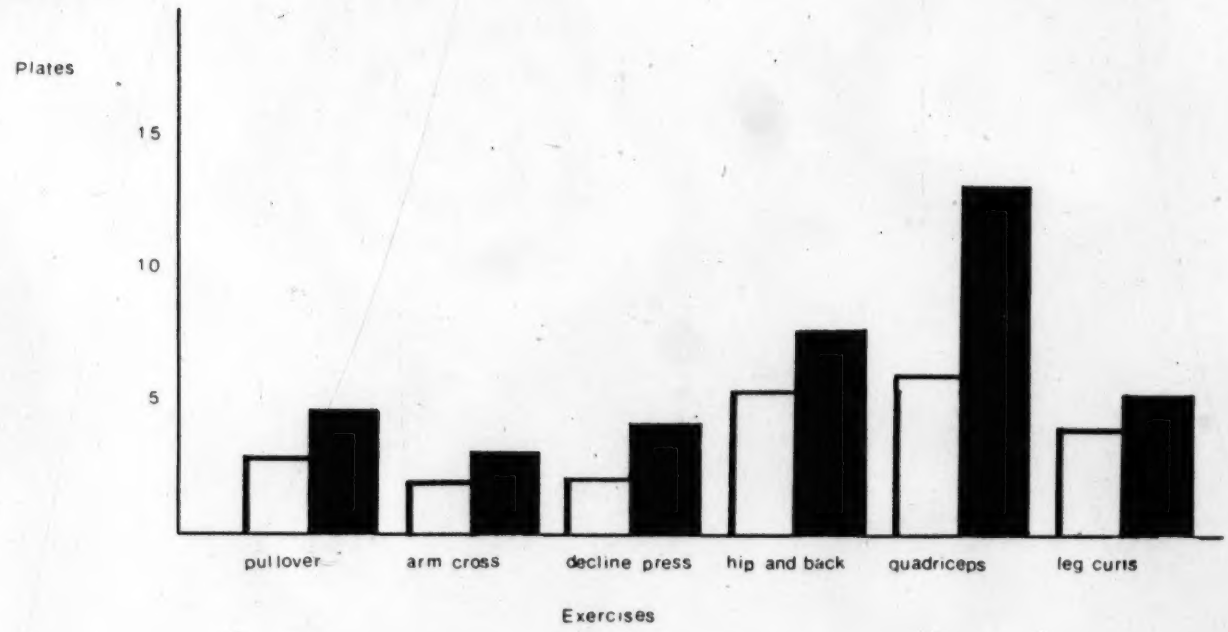

Figure 3 Number of plates for each individual exercise during the pre-test (□) and post-test (■)

TABLE 3: BODY COMPOSITION BEFORE AND AFTER TRAINING

Before Training

Subject	Weight (kg)	Percent Fat	Kilograms Fat	Kilograms Lean
Mean	58.1	24.0	14.2	43.8
SEM	2.3	1.9	1.5	1.3

After Training

Mean	59.3	24.3	14.6	44.7
SEM	3.3	2.1	1.8	2.2

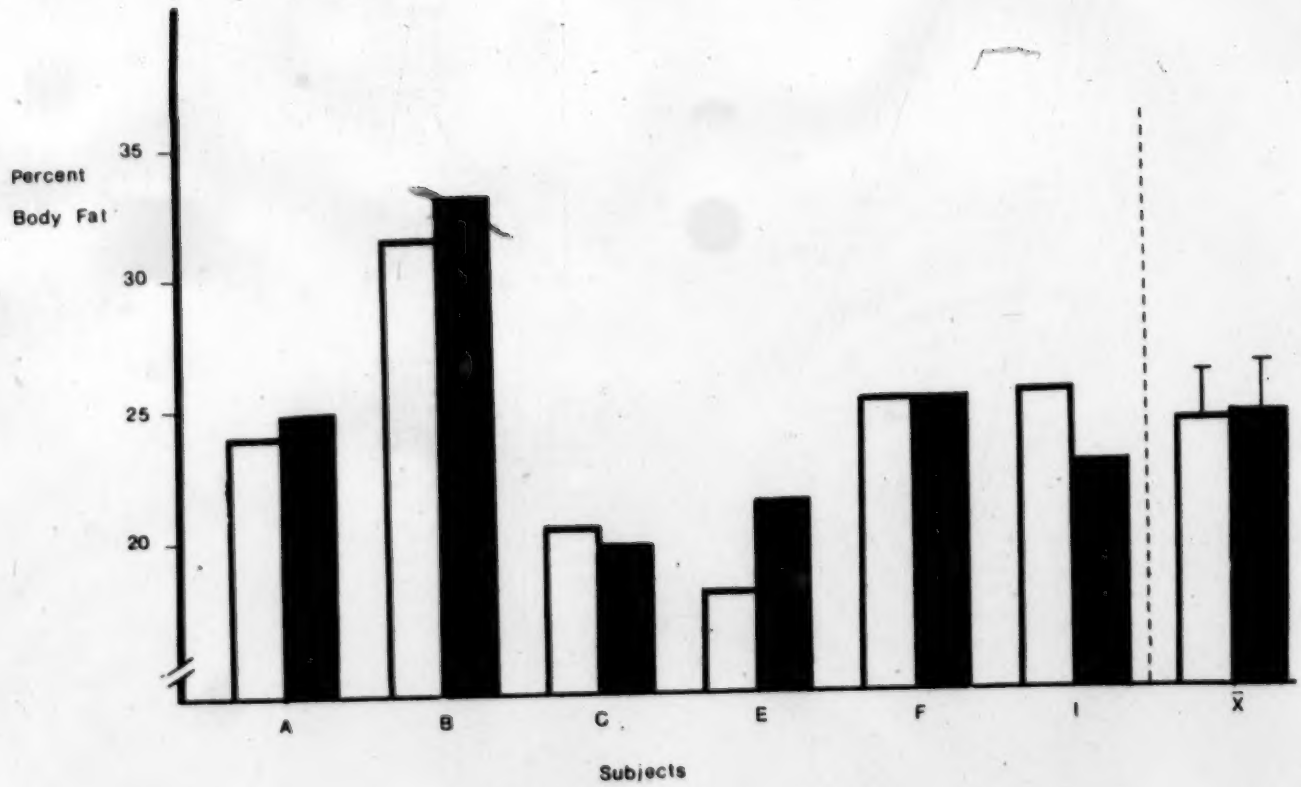

Figure 4: Individual and mean fat compositions of subjects before (□) and after (■) training.

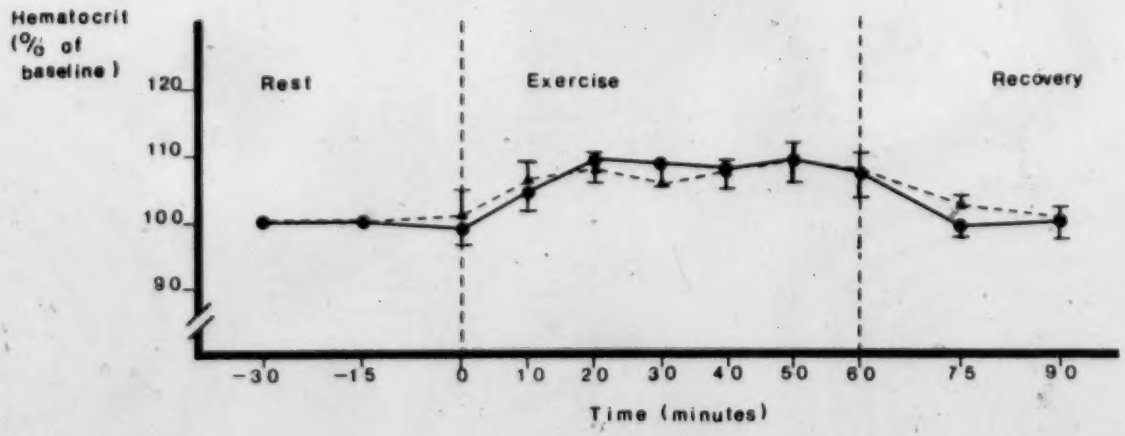

Figure 5. Pre-test (●) and post-test (▲) hematocrit values expressed as a percent of baseline values.

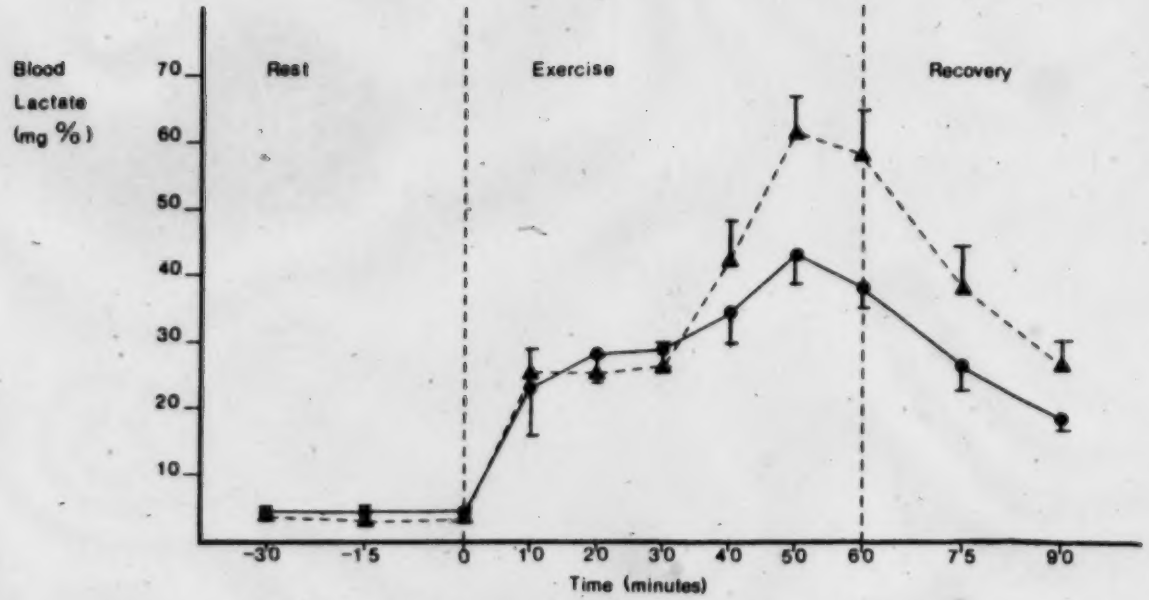

Figure6 · Blood lactate concentrations over rest, exercise, and recovery during the pre-test(●) and post-test(▲).

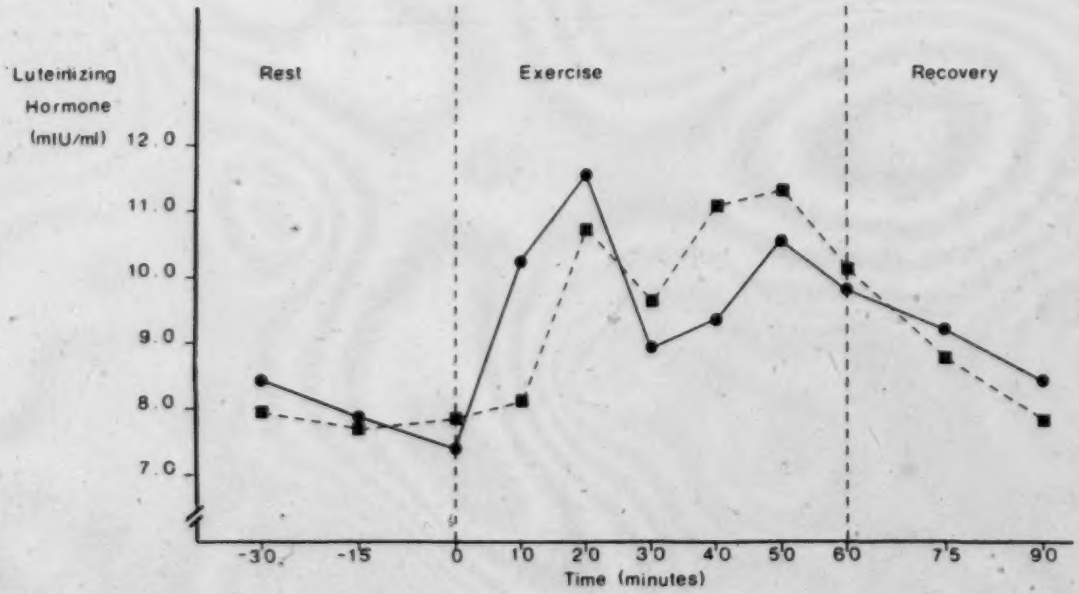

Figure 7. Serum LH concentrations over rest, exercise, and recovery during the pre-test (●) and post-test (■).

TABLE 4: LUTEINIZING HORMONE (mIU/ml)

Pre-Test Samples

	1	2	3	4	5	6	7	8	9	10	11
Mean	8.43	7.88	7.42	10.27	11.56	9.00	9.39	10.56	9.87	9.18	8.44
S.E.M.	1.35	1.15	0.75	1.70	2.42	2.44	1.63	2.04	1.87	1.92	1.43

Post-Test Samples

	1	2	3	4	5	6	7	8	9	10	11
Mean	7.95	7.71	7.84	8.19	10.70	9.64	11.05	11.31	10.12	8.73	7.77
S.E.M.	1.87	1.60	1.64	1.54	1.01	0.96	0.75	1.74	1.34	1.28	0.93

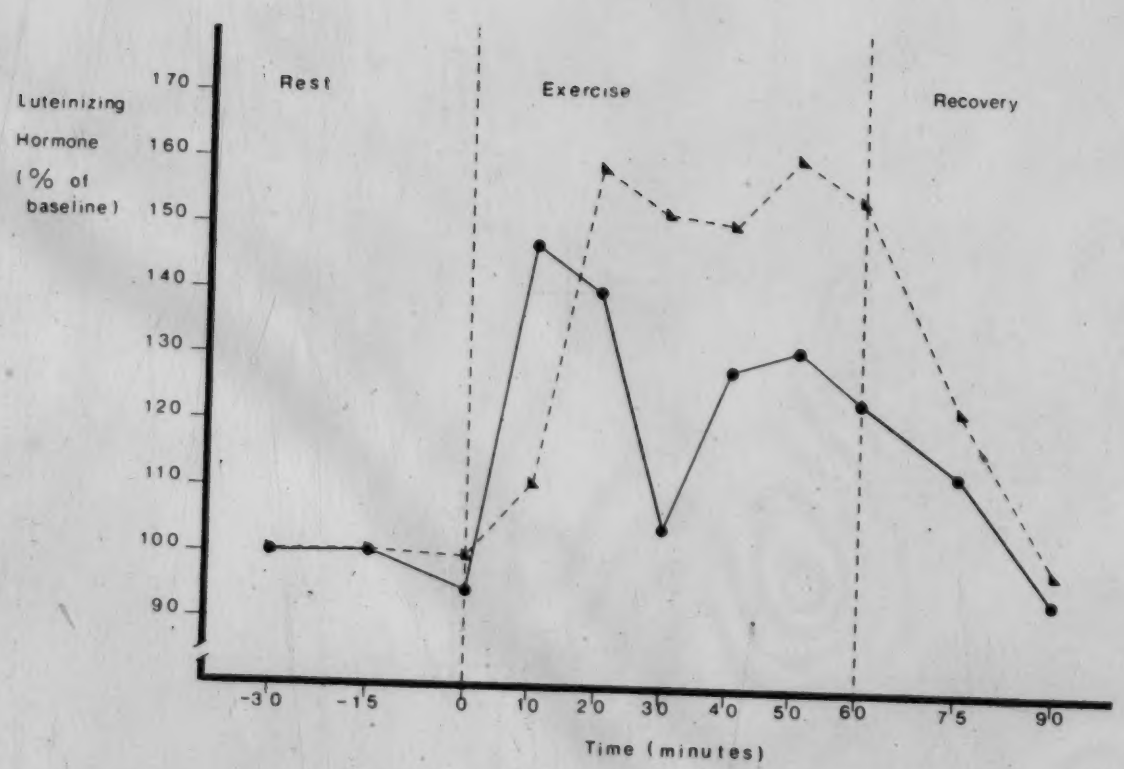

Figure 8 Pre-test(●) and post-test(▲) LH values expressed as a percent of baseline values.

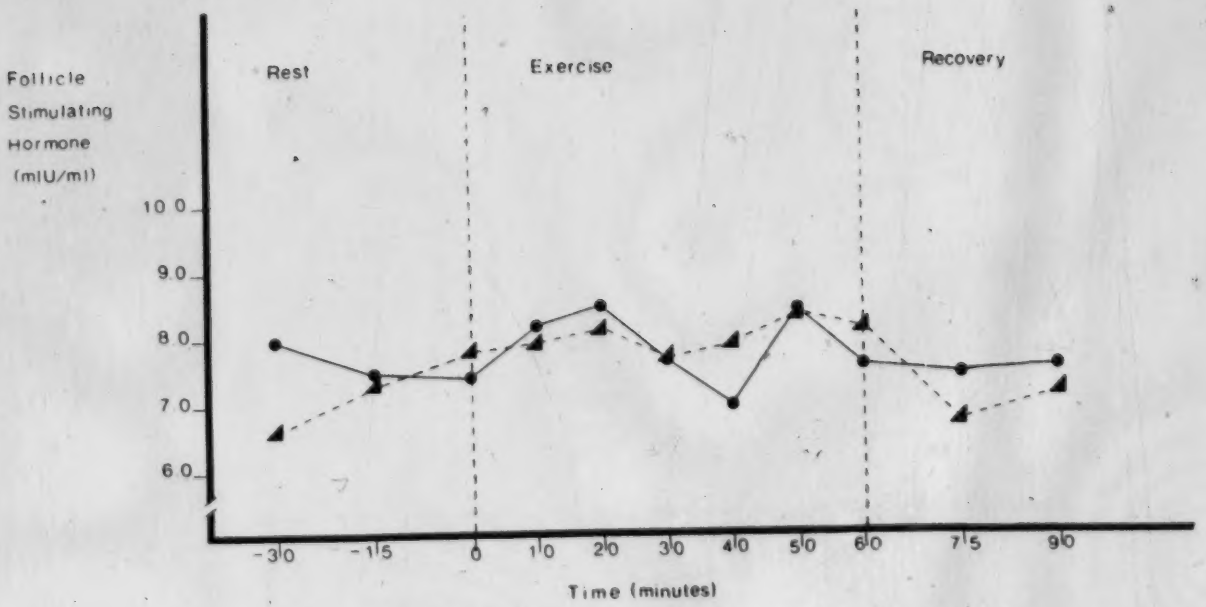

Figure 9 Serum FSH concentrations over rest, exercise, and recovery during the pre-test (●) and post-test (◢)

TABLE 5: FOLLICLE STIMULATING HORMONE (mIU/ml)

Pre-Test Samples

	1	2	3	4	5	6	7	8	9	10	11
Mean	7.95	7.49	7.39	8.17	8.45	7.72	6.93	8.36	7.51	7.39	7.49
S.E.M.	0.79	0.68	0.80	0.57	0.71	0.68	0.56	0.66	0.64	0.61	0.64

Post-Test Samples

	1	2	3	4	5	6	7	8	9	10	11
Mean	6.59	7.27	7.76	7.85	8.02	7.69	7.83	8.26	8.16	6.66	7.06
S.E.M.	1.06	0.69	0.80	0.91	1.29	1.05	1.00	1.95	1.36	1.46	1.82

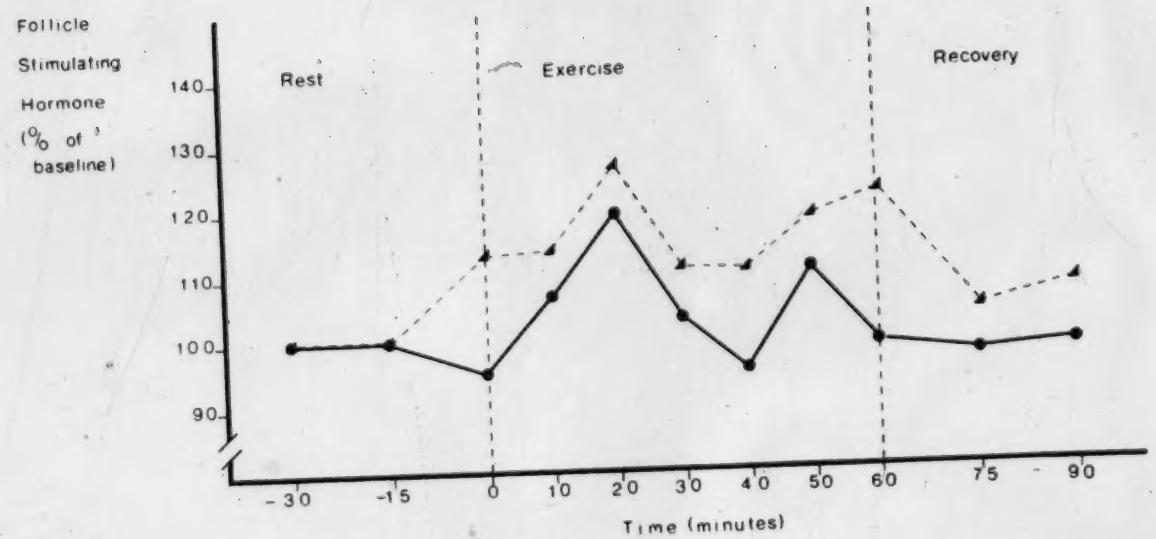

Figure 10 Pre-test (●) and post-test (▲) FSH values expressed as a percent of baseline values

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IV. DISCUSSION

A training effect was demonstrated for all six resistance exercises as evidenced by the increased amount of work performed in the post-test. On the average, the resistance for upper body exercise increased a total of eight plates while that for lower body exercise increased a total of ten plates. The strength gains cannot be expressed in terms of percent increase and thus cannot be equated with values from the literature however, the training program was based on recommendations made by the manufacturers of Nautilus equipment (see appendix E). Furthermore, attainment of "optimum" strength gains was not a contingency upon which the study was based.

The blood lactate results supply further evidence of a training effect. Training usually results in lower blood lactate levels at the same absolute workload and higher blood lactate levels at maximum workloads (Astrand and Rodahl, 1977). In the present study, training resulted in similar blood lactate levels during pre-test and post-test upper body exercises, and higher blood lactate levels during post-test than during pre-test lower body exercises. The higher concentration of lactate in the blood after lower body exercise compared to upper body exercise is indicative of the greater muscle mass involved and the greater amount of work performed.

Serum LH levels increased 47% above pre-exercise values with acute exercise in the pre-test and 60% in the post-test. These levels did not remain elevated but rose and fell twice over the exercise period. Thus, earlier studies which showed no change in LH levels with acute exercise (Dessypris et al., 1976; Bonen et al., 1979) may have in fact missed an increase since they did not analyse serial blood samples throughout exercise. Jurkowski and associates (1978) did analyse serial blood samples and found a small but insignificant rise in LH levels with exercise. It is possible that in this last case the null hypothesis was too readily accepted and that a larger subject population would produce significant results.

The increased LH levels cannot be explained solely by a hemoconcentration effect since hematocrit rose less than 10%. A decrease in metabolic clearance rate may play a role in the LH increase with exercise however there is no conclusive evidence that this is the case. Although renal vascular resistance may increase in exercise, decreasing the proportion of the cardiac output

reaching the kidneys, the cardiac output itself increases (Berne and Levy, 1981). Thus the renal LH clearance may not be significantly affected. Furthermore, the sudden fluctuations in LH concentration are not easily explained by such a gross mechanism. Rather it would appear that increased LH secretion is responsible for the net increase in blood LH levels. Still one can only speculate upon the exercise-associated factors which mediate the increase.

GnRH secretion from the hypothalamus into the hypothalamo-hypophyseal portal system regulates the release of both LH and FSH from pituitary gonadotropes. The exercise-induced enhancement of LH secretion which occurred independent of a change in FSH levels is probably due to an altered pattern of GnRH release since changes in the frequency or amplitude of GnRH have been shown to favour the release of one gonadotropin over the other (Wildt et al., 1981). However, the stimulus for altered GnRH release has yet to be delineated. Catecholamines and/or endogenous opiods may be involved as both have been shown to modulate LH levels, (Barraclough and Wise, 1982; Ropert et al., 1981), and peripheral levels of both have been shown to change with exercise (Terjung, 1979; Carr et al., 1981). The precise effects of these substances on GnRH release (and eventually LH release) are not known and circulating levels do not necessarily reflect synaptic levels, still one can recognize that neural pathways exist by which central nervous system input may modulate GnRH release and this input may be altered in the exercise situation.

In 1981, Cumming and associates reported increases in serum LH levels in women prior to exercise on a bicycle ergometer. LH levels continued to rise as exercise intensity was increased incrementally to a symptom-limited maximum. The pattern of LH response not only illustrated the involvement of neuroendocrine mechanisms in the control of LH secretion, but also the likelihood that cognizance of the physical stress is a fundamental component of the physiological reaction to that stress.

Additional evidence supporting this theory was provided by Cumming and associates as they attempted to dissociate the changes in LH levels due to anticipation from those due to the exercise itself. Subjects who were expecting to exercise but who were not permitted to exercise and who were not informed until 10 minutes after the proposed starting time that they would not be

exercising demonstrated i) anticipatory increases in LH levels similar to those demonstrated by the exercising group and ii) elevated LH levels during the proposed exercise period that were lower but of similar duration to those of the exercising group. In addition, Rebar and associates (1983) found that women who were informed that they would not be exercising did not show an increase.

In the present study no anticipatory rise in LH levels was observed. Possible explanations include the different nature of the work to be performed (Nautilus vs bicycle ergometer, set resistance vs increasing resistance, intermittent vs continuous exercise, anaerobic vs aerobic exercise) and greater subject familiarity with the exercise. If subject familiarity is a factor it may help explain the delayed rise in LH levels seen in the post-test.

Despite the fact that no LH increase was observed in anticipation of exercise, psychological preparatory factors or arousal mechanisms may have been involved in the present study to produce the biphasic LH response. Although each exercise was performed against maximal resistance, one LH peak occurred during upper body exercise and a second peak occurred during lower body exercise. This pattern is difficult to explain in terms of a purely physical stress response. Unlike lactate the response did not appear to be related to muscle mass or the actual amount of work performed as the peak values obtained were not significantly different for upper body or lower body exercise. Rather the response appeared to be related to the order of the two groups of exercises. The LH response did appear to be related to the relative intensity of the exercise in that it was similar for the pre-test and post-test despite differing absolute exercise intensities, thus a form of "endocrine conditioning" may have occurred with training.

The elevated LH levels observed in this study fall well within the normal physiological range for the early follicular phase of the menstrual cycle. Nevertheless, it is possible that regular strenuous physical activity with concomitant elevations in LH levels may be stimulus enough to disrupt menstrual function in susceptible individuals. The sustained increases in LH levels with exercise which were observed in normally-cycling women after training may represent a minor aberration in normal reproductive functioning on a continuim with the more serious aberrations produced in amenorrheic athletes. Exercise-induced modulation of LH secretion may be designed

to discourage procreation in a physiologically inappropriate situation.

Alternatively, the observed increases in LH levels in response to exercise may be physiologically significant in terms of an adaptive response to enhance strength training. LH is the stimulus for androgen secretion from the ovaries and androgens have been implicated in the development of muscular strength and hypertrophy (Lamb, 1975). In fact, a mild hyperandrogenism was demonstrated in these subjects after training (Galbraith et al., 1982). Thus, alterations in menstrual function due to exercise of this type may be secondary to an altered testosterone/estradiol ratio; an endocrine response designed to promote adaptions in muscle.

In conclusion, the results of this study further implicate central nervous system modulation of the hypothalamo-pituitary axis in the LH response to exercise. Further research involving manipulation of the exercise sequence and the analysis of psychological data may eventually provide additional support for this hypothesis.

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VI. APPENDIX A: REVIEW OF LITERATURE

A. THE MENSTRUAL CYCLE

The menstrual cycle of the human female is a repetitive expression of the integrated processes of ovarian steroidogenesis and follicular development, and neuroendocrine control mechanisms. The orderly sequence of endocrinologic and morphologic events which characterize the cycle are the result of complex interplay among the Central Nervous System (CNS), hypothalamus, pituitary, and ovaries. It is the ovarian unit, which by virtue of its follicular compartment is inherently cyclic, that directs the time course of events in the normal cycle (Knobil, 1974).

THE OVARIAN CYCLE

The granulosa cells of developing ovarian follicles are capable of synthesizing all three classes of sex steroids however, significantly more estrogens (>1 pg/cell/48 hrs) than either androgens (<0.05 pg/cell/48 hrs) or progestins (0.7± 0.3 pg/cell/48 hrs)(McNatty et al., 1979b). The factor limiting follicular estrogen production is the aromatase enzyme system which converts androgens to estrogens. The aromatization process is induced by follicle-stimulating hormone (FSH) from the anterior pituitary thus, FSH and FSH receptor content are limiting factors in early follicular estrogen production (Moon et al., 1975). FSH stimulation will induce an increase in the number of FSH receptors plus act synergistically with estrogen to enhance mitogenic activity of the granulosa cells causing proliferation (Richards, 1979). The result is an increased number of granulosa cells and an increased receptor density on those cells. FSH attaches to FSH receptors on the granulosa cells, converts androgens to estrogen, and generates the estrogenic microenvironment considered essential for continued follicular growth (Fritz and Speroff, 1982).

As well as providing a substrate for FSH-induced aromatization to estrogen, androgens may have a further role in enhancing aromatase activity. Specific androgen receptors are present in the cytoplasm of granulosa cells (Schreiber and Ross, 1976) and granulosa cell aromatase activation by FSH has been shown to be an androgen receptor regulated process in vitro (Hillier and DeZwart, 1981). It appears that aromatization and estrogen production are enhanced by low

androgen levels but that the limited capacity for aromatization may be saturated by higher androgen levels resulting in an androgenic and ultimately atretic follicle (Hillier et al., 1980). Thus, it is possible that only those follicles emerging in late luteal phase or early follicular phase when FSH is elevated will proceed in development.

With continued growth each follicle develops an antrum filled with follicular fluid and an endocrine environment unique to itself. Theca cells add to the follicular estrogen production by elaborating androgens for aromatization by granulosa cells (McNatty et al., 1979a). This co-operative effort has been termed the two-cell, two-gonadotropin concept. Androstenedione produced by thecal cells under luteinizing hormone (LH) stimulation is the primary substrate for 17β-estradiol synthesis by granulosa cells under FSH stimulation (Tsang et al., 1980). Thus, accelerated estradiol production may occur later in antral development, manifest in the estradiol surge.

Selection of the follicle destined to ovulate may also be contingent upon the interaction between estradiol and FSH. While estradiol has a positive influence on FSH action within the maturing follicle it also has a negative feedback influence on FSH release from the pituitary. Therefore, while a dominant follicle, with a highly proliferate granulosa and numerous FSH receptors, may be able to retain unique FSH responsiveness and continue to develop despite a drop in FSH levels, FSH support will be withdrawn from less developed follicles (Zeleznik, 1981). These follicles will experience a decline in aromatase activity, become androgenic and finally atretic.

A second ovarian product that may be involved in the selection process is the protein hormone inhibin. Inhibin is synthesized by granulosa cells and secreted into the follicular fluid and ovarian venous blood (Marder et al., 1977). It appears to suppress pituitary FSH secretion and thus, like estradiol feedback, to promote atresia.

The negative feedback effect of estradiol on FSH release from the pituitary is an almost immediate response even at low levels. However, estradiol influence on LH release varies with concentration and the duration of exposure (Knobil, 1974). Negative feedback is present at all

estradiol concentrations but positive feedback has an additional role at high estradiol concentrations. As estradiol rises during the midfollicular phase of the cycle the balance shifts from suppression to stimulation of LH release. An LH surge will result if threshold estradiol values of 200 pg/ml are reached and maintained until after the LH surge has begun (Young and Jaffe, 1976).

FSH has been implicated in the induction of LH receptors on the granulosa cells of large antral follicles (Zeleznik et al., 1974). The rate of appearance of LH receptors has been shown to increase with increased exposure to estradiol (Richards and Midgley, 1976). Therefore, not only does accelerated estradiol production act centrally to stimulate the LH surge, but it acts locally as well to promote induction of the LH receptors required for response to the surge.

Thirty-six to fourty-eight hours prior to ovulation plasma estradiol is at a zenith. The sustained threshold estradiol concentration stimulates the LH surge. LH binds to specific receptors on granulosa cells, promotes luteinization and progesterone production. There is evidence to suggest that the preovulatory rise in progesterone that results is necessary for the midcycle FSH surge and augments the midcycle LH surge (March et al., 1979).

The LH surge appears to trigger ovulation. Increased LH levels initiate resumption of meiosis, luteinization of the granulosa, and synthesis of prostaglandins essential for follicle rupture (Tsafriri et al., 1972). Increased follicular levels of cyclic adenosine 3':5' monophosphate appear to mediate LH activity by overcoming local inhibition by oocyte maturation inhibitor (OMI) and luteinization inhibitor (LI)(Tsafriri et al., 1976). There is evidence that progesterone, as well as prostaglandins, are involved in follicle rupture (Bjersing, 1979).

While the first half of the reproductive cycle is governed by the developing follicle, the second half is governed by the corpus luteum. Yet normal luteal function is only possible after optimal preovulatory follicular development. The functional capacity of the corpus luteum is pre-determined by the extent of luteinization which likewise depends on the accumulation of LH receptors during the follicular phase and LH secretion (Stouffer and Hodgen, 1980).

The corpus luteum is the principle source of luteal phase progesterone. Progesterone levels increase rapidly after ovulation reaching a peak approximately eight days after the LH surge. Progesterone acts to suppress new follicle growth by central inhibition of FSH release from the pituitary (diZerega and Hodgen, 1980) and local inhibition of folliculogenesis at the ovary (Schreiber et al., 1980).

Progesterone levels decline gradually to basal levels after the peak. The corpus luteum becomes less sensitive to LH stimulation with age and decreases in steroidogenic capacity (Stouffer et al., 1977). This may be due to the increasing concentration of LH receptor binding inhibitor (LHRBI) in luteal tissue and/or an estradiol-induced decrease in LH receptor binding capacity (Sotrel et al., 1981). Estradiol was originally implicated in luteolysis as the midluteal rise in estradiol occured coincident with the decline in progesterone. It has since been shown to induce luteolysis upon parenteral administration (Karsch and Sutton, 1976). Prostaglandins appear to mediate the luteolytic action of estradiol as inhibitors of prostaglandin synthesis block the effect (Auletta et al., 1978).

NEUROENDOCRINE CONTROL OF GONADOTROPIN SECRETION

Rhythmic pulses of FSH and LH are superimposed on low level continuous secretion. This pulsatile pattern is not intrinsic to the pituitary but rather a reflection of intermittent hypothalamic stimulation. The hypothalamus integrates neural input from the CNS with feedback signals from the ovary, and modulates gonadotropin secretion through release of gonadotropin releasing hormone (GnRH).

Diverse neural inputs converge on GnRH neurons concentrated primarily in the arcuate nucleus of the medial basal hypothalamus (Silverman et al., 1977). Their neurotransmitters modulate the release of GnRH pulses from nerve terminals in the median eminence, into the portal system to the anterior pituitary. GnRH binds to specific GnRH receptors on the pituitary gonadotropes to effect the release of the gonadotropins into the systemic circulation.

While GnRH is the single releasing hormone common to LH and FSH (Kao et al., 1977), the pattern of LH and FSH release may vary as a result of feedback modulation by gonadal hormones at the hypothalamus-pituitary axis (Rebar and Yen, 1979). The frequency and magnitude of gonadotropin pulses varies with the phase of the cycle (Yen et al., 1972). Throughout most of the cycle pulses occur every hour or hour and a half. During the mid and late luteal phase the frequency drops to every three or four hours. It is possible that progesterone has a dampening effect on the central neuronal mechanisms for gonadotropin release and is thus responsible for the decreased frequency in the luteal phase (Rebar and Yen, 1979). Pulse amplitude is generally lowest during the late follicular phase and highest during the midcycle surge. There is evidence to suggest that increasing levels of circulating estradiol, through feedback modulation, are responsible for the change observed between early and late follicular phase (Yen et al., 1972).

Evidence suggests that both estradiol and progesterone exert feedback effects at both the hypothalamus and pituitary levels. Estradiol receptors have been demonstrated in the anterior pituitary (Pfaff et al., 1976) and pituitary responsiveness to GnRH is influenced by duration and concentration of estradiol (Yen et al., 1974). Knobil (1980) suggested that estradiol could influence pituitary sensitivity to GnRH by altering the GnRH receptor content however, it has since been demonstrated that pituitary sensitivity to GnRH is not always correlated with receptor content (Adams and Spies, 1981; Ferland et al., 1981). Pituitary sensitivity to GnRH is only positively correlated with receptor content when positive estradiol feedback is in effect. Negative estradiol feedback appears to operate through a different mechanism, possibly by exerting inhibitory effects in both the hypothalamus and pituitary (Adams et al.,1981). Recent evidence suggests that progesterone acts on the hypothalamus to inhibit gonadotropin release but on the pituitary to facilitate the midcycle gonadotropin surge (Wildt et al., 1981). Finally, feedback of steroids above the levels of the pituitary may modulate the pattern of GnRH pulses, the significance of which has only recently been demonstrated. Wildt and associates (1981) have shown that changes in the frequency or amplitude of GnRH pulses not only affect gonadotropin levels but the relative ratios of LH and FSH as well.

Central nervous system input may override the interacting elements of hypothalamus, pituitary and ovaries to alter the established pattern of reproductive functioning. Aminergic neurons from the CNS have been demonstrated to interact with the GnRH neurons of the hypothalamus and influence GnRH release. Modulating this interaction are other neurotransmitters, endogenous opiods, gonadal steroids and prostaglandins.

Morphological (Fuxe et al., 1976) and immunohistochemical (McNeill and Sladek, 1978) evidence demonstrates norepinephrine and dopamine neurons in close contact with GnRH nerve terminals in the median eminence. The bulk of the evidence suggests that norepinephrine plays a stimulatory,and dopamine a inhibitory, role in GnRH release and that GnRH release will reflect the ratio of the two catecholamines (Drouva and Gallo, 1976; Chiocchio et al., 1976; Leblanc et al., 1976; Judd et al., 1978).

Estradiol receptors have been demonstrated in the cell bodies of arcuate dopaminergic neurons (Grant and Stumpf, 1973). There is evidence to suggest that estradiol feedback may involve influencing GnRH release through action on dopaminergic neurons (Yen, 1979; Fuxe et al., 1979). Prolactin, which has been shown to stimulate dopamine neuronal activity in the median eminence (Fuxe et al., 1979) may also cause decreased GnRH release.

Endogenous opiods have been implicated in the regulation of gonadotropin secretion at various stages of the menstrual cycle (Quigley and Yen, 1980). Opiate-receptor blockade results in increased frequency and amplitude of LH pulses during the mid-luteal phase (Ropert et al., 1981). No similar effect was observed during the follicular phase (Quigley and Yen, 1980) indicating that ovarian steroid feedback may modulate endorphin inhibition of GnRH.

B. ADIPOSE TISSUE

FAT CELL SIZE AND NUMBER

Much controversy exists concerning the relationship between size or number of adipocytes and the degree of adiposity in adults. Evidence that i) overfeeding rats in the first few weeks of life leads to permanent obesity and an increase in fat cell number while ii) later nutritional manipulation affects adipose tissue mass by increasing cell size only (Knittle and Hirsch, 1968; Hirsch and Han, 1969) suggests that the hyperplastic period is finite. This, coupled with findings that adults with childhood-onset obesity have a greater number of fat cells than those with adult-onset obesity (Hirsch and Knittle, 1970; Knittle, 1972; Salans et al., 1973), gave rise to the "fat cell hypothesis", that overfeeding in the hyperplastic period causes development of excess fat cells which predispose a person to obesity (Pawan, 1971).

The prospect of a finite period of hyperplasia is not unique to adipose tissue. Studies on rats, guinea pigs and some human tissues have demonstrated one pattern of growth common to all non-regenerating organs (Kirtland and Gurr, 1979). The pattern involves an initial period of hyperplasia, a period of combined hyperplasia and hypertrophy, and a final period of hypertrophy alone. The duration of the hyperplastic period varies among organs and among species but is generally confined to the early life of the animal. For human adipose tissue it is thought to encompass the period from age thirty weeks of intrauterine life up to one year of extrauterine life (Brook et al., 1972).

Extrapolation from rat data may not be justified since humans, unlike rodents, have a development pattern characterized by two periods of rapid growth; one in early life and one at puberty (Hirsch and Batchelor, 1976). Secondly, fat distribution in rats is different from that in humans (Greenwood and Johnson, 1977) and much of the rat data has been obtained from experimentation on epididymal fat pads (not present in humans) while subcutaneous fat is most often studied in humans. Several researchers have suggested the existence of one sensitive period in the first year of life and another in early adolescence (Brook, 1975; Salans et al., 1973;

Abebonojo, 1975) implying that there is no one finite period of hyperplasia for adipose tissue.

The theory that early-onset obesity is a pathological consequence of hypercellularity imposed during the hyperplastic period(s) has since been refuted (Chumlea et al., 1981). Individuals with early-onset obesity and little or no hypercellularity have been found, as have individuals with late-onset obesity and marked hypercellularity (Hirsch and Batchelor, 1976). Recent work has demonstrated that fat cell number is only related to the severity of the obesity (Sjostrom and Bjorntorp, 1974; Gurr et al., 1978). Thus, a more appropriate hypothesis may be that overfeeding at any stage leads to hypertrophy of existing fat cells but once these have reached their maximum size (0.8-1.0 ug) any further increase in adipose tissue mass is accomplished by an increase in observable fat cell number. This increase may be due to hyperplasia or, more probably, to "filling of empty cells", bringing them into the size range in which they are observable, which is in fact hypertrophy. Thus, acquisition of excess observable fat cells would be a consequence, and not a cause, of massive obesity (Kirtland and Gurr, 1979). Finally, errors inherent in the techniques presently available for the determination of fat cell number should be considered when evaluating conclusions drawn from the literature (Gurr and Kirtland, 1978).

ENERGY METABOLISM

Adipose tissue is highly active tissue specifically adapted for the alternate functions of storage and mobilization of fat as a fuel for energy production. By virtue of possessing enzymes for various metabolic pathways, it is capable of synthesizing triglycerides from fat and carbohydrate sources and storing them during periods of positive energy balance. Likewise, in the event of a negative energy balance it is capable of hydrolyzing triglycerides to release fatty acids into the bloodstream. The ability to regulate flux through such essential metabolic pathways is dependent on the ability to rapidly modify the catalytic activity of the enzymes in these pathways. Various mechanisms exist to achieve this short-term regulation, some mediated by hormones and others by changes in the concentration of certain metabolites within the cell compartments. These metabolic pathways are in balance between the anabolic effects of insulin and the catabolic effects of a

number of other hormones, such as glucagon and the catecholamines.

The anabolic effect of insulin is manifest in enhanced triglyceride synthesis and storage, and inhibited lipolysis. Insulin enhances lipoprotein lipase activity thus enhancing fatty acid uptake by adipocytes (Newsholme and Start, 1979). Insulin is required for the facilitated diffusion of glucose into fat cells, stimulates glycolysis and α-glycerophosphate production, and prompts triglyceride storage (Saggerson, 1980). These actions may simply be the result of increased substrate availability however, there is evidence to suggest that insulin may directly stimulate a number of pathway enzymes including hexokinase, the pyruvate dehydrogenase complex, and acetyl-coA carboxylase (Denton, 1977). The mechanism(s) by which insulin exerts its antilipolytic action is equally unclear. Possibilities include the inhibition of adenylate cyclase (D'Costa et al., 1979), stimulation of cAMP phosphodiesterase (Fain et al., 1978) and inhibition of cAMP-dependent protein kinase (Pohl et al., 1981).

The actions of glucagon, adrenocorticotrophic hormone (ACTH), and the catecholamines oppose those of insulin. These hormones stimulate lipolysis by binding to specific surface receptors and increasing adenylate cyclase activity. Thus, they increase cAMP levels, activating cAMP-dependent protein kinase which phosphorylates and activates triacyglycerol lipase (Galton and Stansbie, 1981).

The permissive roles played by growth hormone and glucocorticoids are unclear although they appear to increase the sensitivity of fat cells to lipolytic agents such as catecholamines (Fain et al., 1978). A one to two hour lag period is required for expression of the lipolysis-enhancing action of both hormones, and protein synthesis appears to be involved (Fain, 1973). Likewise, the mechanism behind the accelerated lipolysis which follows four to six hours after thyroid hormone administration has not been delineated. None of these three hormones are considered to alter catecholamine binding on adipocytes (Fain et al., 1978).

Adipose tissue plays a fundamental role in adaption to physiological situations characterized by a negative energy balance (e.g. exercise training, food restriction, cold acclimitization, hyperthyroidism), responding with enhanced lipolytic capacity. Yet the

biochemical mechanisms behind the enhanced lipolytic capacity and the nature of the rate-limiting metabolic steps are unclear. Recent evidence suggests that the lipolytic effects of catecholamines may be increased by exercise training or food restriction at a metabolic step distal to stimulus recognition by receptors, possibly at the level of protein kinases or lipases (Bukowiecki et al., 1980).

C. STRENGTH TRAINING

PRINCIPLES OF STRENGTH TRAINING

The main principles of strength training are well acknowledged. To gain strength through training it is necessary to load muscles beyond the point to which they are normally loaded. As muscles become stronger they must work against a progressively greater resistance (Wilmore, 1977). However, the optimum strength training program has not yet been determined. Interpretation of comparative studies is difficult due to a lack of standardization (Ramos, 1976), and the bias imposed by the choice of strength-testing methodology (Pipes, 1978). Research in this area is further complicated by the large number of interrelated variables that are manipulated in the establishment of any given training program. For example, it has long been acknowledged that heavy resistance, low repetition training improves strength while light resistance, high repetition training improves endurance, yet the true independent load-gain and frequency-gain relationships are left unresolved since increasing the load necessarily requires decreasing the frequency.

In isotonic training submaximal loads are generally considered to produce the best results. Programs incorporating 5-6 RM loads (ie. loads that can be lifted a maximum of five or six times), three sets a session, three sessions a week are supposed to produce near optimal results (Wilmore, 1977; Atha, 1981). Speed loading is not recommended as it increases the effective load on the muscle during the acceleration phase only, after which time the load intensity and thus the training stimulus is in fact decreased (Atha, 1981). However, new apparatus have been developed which allow more complete isokinetic loading throughout the entire range even at high speeds. The effect of introducing an inter-repetition rest interval has not been investigated although logically this factor should influence the rate of fatigue and the training stimulus (Atha, 1981).

Variable resistance training may provide some benefits over the standard constant-resistance training. Theoretically, variable-resistance training should enhance strength gains by producing a relatively uniform stress over the entire range of motion, eliminating the tendency for failure to occur only at the weakest point in the range of motion (Coleman, 1977;

Heusner, 1981). Experimental evidence to this effect is so far lacking. Present evidence indicates that strength gains through variable resistance training are superior to those obtained through constant resistance training only at the joint angle at which peak loading is induced (Atha, 1981) or when measured on variable resistance apparatus (Pipes and Wilmore, 1975).

Maximal contractions are considered optimal in isometric training (Wilmore, 1977; Atha, 1981). Duration of contraction should be long enough to allow full recruitment of muscle fibers (Atha, 1981), a property which may vary among muscles (Wilkie, 1966). One second maximal contractions have been shown to increase strength (Atha, 1981) while increasing durations produce diminishing benefits (Muller, 1970) and very long contractions may in fact be counterproductive (McGlynn, 1968). Repeated contractions appear to be beneficial (Muller, 1970) although neither the optimum number of repetitions nor an appropriate rest-interval have been determined. Daily training has consistently proved superior to less frequent training (Atha, 1981) while twice daily training may in the long run be deleterious (McGlynn, 1968). Finally, training at more than one joint angle may be beneficial since strength gained from training at one joint angle may not always be transferable to other angles (Williams et al., 1978).

The bulk of the evidence in isokinetic training recommends moderately slow-speed training over either ultra-slow or high-speed training (Atha,1981). However, the possibility remains that fast-speed training may be more effective in developing the strength needed for fast, powerful movements (Perrine and Edgerton, 1975). The benefits of increasing the frequency of repetitions may be limited, as indicated by preliminary papers (Lesmes et al., 1978). No information has been found to support a specific number of sets per session or sessions per week over any others.

THEORIES OF STRENGTH GAINS

It is well acknowledged that the magnitude of the strength gain attainable by training diminishes as training status improves (Wilmore, 1977). The adaptions resulting from training which allow a muscle to approach its theoretical maximum in terms of force production are not

perfectly clear. Several factors have been implicated.

Muscle hypertrophy has long been considered a prerequisite for increased muscle strength. Usually bigger muscles can produce greater force output (Komi, 1979). However, strength increases are not always significantly correlated with increases in muscle mass (Wilmore, 1977), particularly in females. It appears that hypertrophy, while sometimes a by product of strength training, is not a necessary consequence (Wilmore, 1974; Lesmes et al., 1978). Other factors such as improved innervation and strengthening of the muscle itself may be more important (Komi, 1979).

Muller (1970) suggested that specific coordination patterns may be "learned" with training, enhancing force output. Training may instill the ability to partially overcome inherent autogenic inhibition and thus allow greater stimulation of muscle contraction (Wilmore, 1977). Lesmes and associates (1978) hypothesized that much of the observed increase in strength may be explained on the basis of adaptions in the neurological control of muscle fibre recruitment. Training may increase the number and/or nature of fibers recruited and/or cause more synchronous firing.

Muscle fiber composition is known to influence mechanical aspects of muscle function. Fast twitch fibers can produce higher peak velocities and greater force output at a given contraction velocity than slow twitch fibers and are characteristically recruited during high intensity exercise (Komi, 1979). Training may not only influence the motor unit recruitment pattern (Tesch and Karlsson, 1978) but the metabolic profile of the muscle fibers as well (Jansson et al., 1978).

D. EXERCISE-RELATED MENSTRUAL DYSFUNCTION AND ASSOCIATED FACTORS

Menstrual dysfunction is prevalent among female athletes. Delayed menarche may result from intense training in the pre-menarcheal years (Frisch et al., 1981) and reports of exercise-related oligomenorrhea and amenorrhea are on the increase. While this increase is probably a consequence of increased female participation in athletics it may also reflect an increased awareness of the condition (Cumming and Belcastro, 1982).

It is not known what physiological consequence(s) of training is/are conducive to amenorrhea. One or a combination of factors may be involved to upset the intricate sequence of inter-dependent events within the hypothalamus-pituitary-ovarian axis and disrupt menstrual functioning. A number of endogenous personal factors have also been suggested to increase the susceptibility of some women to secondary amenorrhea. These factors include age (chronological, menarcheal, gynecological) (Baker et al., 1981; Speroff and Redwine, 1980; Warren, 1980; Frisch et al., 1981; Shangold and Levine, 1982), gravidity (Baker et al., 1981; Schwartz et al., 1981) and prior menstrual irregularity (Schwartz et al., 1981; Shangold and Levine, 1982). Thus, it is possible that hypothalamus-pituitary-ovarian axis "immaturity" may predispose some women to the deleterious effects of exercise training on reproductive function.

The nature, intensity, and duration of physical activity appear influential in the developmment of oligomenorrhea and amenorrhea. Erdelyi (1976) determined that oligomenorrhea and amenorrhea were most frequent among athletes participating in sports requiring strenuous physical activity. He cited tennis, rowing and skiing as examples. Dale and associates (1979), surveying competitors in a marathon, observed that "the severity of physical exertion, as judged by the mileage run per week, is directly proportional to the degree and incidence of menstrual dysfunction". Extrapolating from data on college cross-country runners, 6% to 43% of whom were amenorrheic depending on their weekly mileage, Feicht and associates (1978) predicted that 50% of marathon runners and national calibre distance runners might be expected to have amenorrhea.

While Sanborn and associates (1982) also found that the frequency of secondary amenorrhea in runners increased with weekly training mileage, no similar relationship was demonstrated for either swimmers or cyclists. The authors suggest that this discrepancy may be explained by the fact that runners, but not swimmers or cyclists, show a weight loss parallel to the increase in training mileage.

Low body weight, weight loss, and low percent body fat are factors implicated in the development of secondary amenorrhea as body composition has long been considered an important factor in the maintenance of regular cycles. Since metabolism of steroid hormones occurs in fat tissue, an alteration in fat weight may alter the hormonal milieu and ultimately steroid feedback at the level of the hypothalamus and pituitary (Fishman et al., 1975; Wentz, 1980). Frisch and McArthur (1974) suggest that a critical lean/fat ratio is necessary for menarche and the continuation of menses. Cessation of menses may result from simple weight loss (Wentz,1980), or weight loss due to anorexia nervosa (Fries, 1974) but may return with normalization of body weight (Knuth et al., 1977). Similarly, cessation of menses with weight loss and resumption of menses with weight gain have been noted in athletes (Frisch et al., 1981).

According to Speroff and Redwine (1980) weight and weight loss are the predominant factors in the development of oligomenorrhea and amenorrhea. Accordingly, Frisch and associates (1980), using a weight for height nomogram to estimate body fat, found that amenorrheic ballet dancers were significantly leaner than normally-cycling dancers. Shangold and Levine (1982) found that amenorrheic marathon runners had significantly lower weight/height ratios and Schwartz and associates (1981), using skinfold measurements, found that amenorrheic runners were leaner than normally-cycling runners and both groups were leaner than sedentary controls. In contrast, Feicht and associates (1978) found no difference between amenorrheic and menstrually regular middle-distance runners with regard to weight and height and Baker and associates (1981) found no significant difference between amenorrheic and menstrually-regular runners on the basis of skinfold measurements. Moreover, Warren (1980) noted resumption of menses independent of any weight change in ballet dancers during a training hiatus. Finally, in direct opposition to the

findings of Sanborn and associates (1982), that runners have a higher incidence of amenorrhea than do swimmers (possibly a consequence of lower body fat), Frisch and associates (1981) found a higher incidence of amenorrhea in swimmers than in runners and postulated that this may be due to the larger proportion of swimmers who began their training premenarcheally.

Several authors support the view that women who train prior to menarche later experience a higher incidence of menstrual disorders (Erdelyi, 1962; Feicht et al., 1978). In one study by Frisch and associates (1981) 61% of the premenarche-trained athletes had oligomenorrhea and 22% were amenorrheic while only 40% of the postmenarche-trained athletes had oligomenorrhea and none were amenorrheic.

Prior irregularity may predispose a woman to exercise-associated amenorrhea. Schwartz and associates (1981) determined that amenorrheic runners had a higher incidence of prior menstrual irregularity than did normally-cycling runners or non-runners. Shangold and Levine (1982) agreed that a woman's menstrual pattern during training was related to her pattern before training. They found that only 7% of the women whose periods were regular prior to training developed oligomenorrhea or amenorrhea with training while the other 93% remained regular. They also found that 25% of the women who were irregular prior to training became regular with training while the other 75% remained irregular. Thus a beneficial influence of exercise was also demonstrated casting doubt on the hypothesis that strenuous training is the solitary cause of menstrual dysfunction. These authors further speculated that some factor which promotes menstrual irregularity may inspire some women to undertake running. Psychological factors such as perceived stress, anxiety, and ambition were suggested to account for this tendency (Shangold and Levine, 1982). Exercise could be a form of therapy in treating "stress" (Anderson, 1978).

Psychological factors constitute one of the most frequent causes of secondary amenorrhea(Thorn, 1977). Fear, anxiety, and grief are frequent causes of temporary amenorrhea (Thorn, 1977). Disruption of menstruation by travel abroad, imprisonment, fear of pregnancy, sexual problems, exam tension, familial or occupational stresses have all been demonstrated (Keller,1981). According to Warren (1980) "the observation that athletes who are top performers

and participate in sports requiring tremendous physical effort and endurance have a higher incidence of amenorrhea raised the possibility of a stress-related phenomenon".

Amenorrheic runners subjectively associated more stress with their running than did normally-cycling runners (Schwartz et al., 1981). The fact that both groups trained at a similar intensity and thus there was no difference in "physical stress" suggests a role for psychogenic factors in exercise-associated amenorrhea. However, the role of "stress" is difficult to evaluate as is the distinction between psychological and physical stress factors. Seventy-five percent of all female recruits did not menstruate during their first month at the U.S. Military Academy but this proportion dropped to 8% by 15 months time (Anderson, 1979). While the relative physical stress may have been greatest during the first month it is also interesting to note that the pattern of decreasing incidence of menstrual dysfunction is not unlike that seen in concentration camp internees despite continued privation and danger (Wentz, 1978).

It appears likely that body fat and stress are two factors that vary in importance among individuals and each can dispose a woman to menstrual dysfunction through alterations exerted at the hypothalamus-pituitary-ovarian axis. Individual susceptibility may be a consequence of maturity at this axis.

The secretion of gonadotropin-releasing hormone (GnRH) represents a central neuroendocrine control point in the regulation of the menstrual cycle (Speroff, 1981). The quantity of GnRH released must be within a critical normal range and discharged in an appropriate pulsatile fashion for ovulatory menstrual function (Knobil et al., 1980). GnRH is released into the hypothalamic-hypophyseal portal system and as such is not easily quantified however, indirect evidence (i.e. the change in reproductive hormones) relates secondary amenorrhea to hypothalamic suppression (Baker et al., 1981). Hypothalamic suppression may result from alterations in steroid feedback at this level or a change in CNS modulatory control via neurotransmitters.

Elucidation of the fluctuating pattern of reproductive hormone levels that occurs with acute exercise, and the more prolonged changes that occur as a result of chronic exercise, is necessary to determine the etiology of exercise-associated amenorrhea. It has been demonstrated in

normally-cycling women that levels of reproductive hormones are altered in association with acute exercise (Cumming et al., 1981). However, a clear endocrinological picture has not been obtained due to the various groups of subjects tested, exercise protocols and testing methodologies employed, and the phase of the menstrual cycle at testing. Estradiol and FSH have been shown to increase over exercise in the follicular phase of the cycle (Jurkowski et al., 1978). Estradiol and progesterone increased during luteal phase exercise bouts (Jurkowski et al., 1978; Bonen et al., 1979) and progesterone increased during exercise at menses (Bonen et al., 1979). Cumming and associates (1981) tested subjects during the early follicular phase and found that estradiol increased in anticipation of exercise in runners but not until exercise had commenced in non-runners. LH and testosterone rose in anticipation of exercise in both groups and continued to rise to a peak in late exercise.

A hypoestrogenic-hypogonadotropic profile has been suggested for amenorrheic athletes. Estradiol levels have been shown to be suppressed (Dale et al., 1979; Baker et al., 1981) and the estrone/estradiol ratio increased (Schwartz et al., 1981). LH (Dale et al., 1979; Baker et al., 1981) and FSH (Dale et al., 1979) were shown to be in the low to low-normal range. However, Schwartz and associates (1981) found LH to be higher in amenorrheic runners than in normally-cycling runners in the early follicular phase of their cycle. Cumming and associates (1981) found comparable basal LH and FSH levels in amenorrheic runners, normally-cycling runners, and non-runners. However, their amenorrheic runners, while demonstrating an LH rise in anticipation of exercise comparable to that seen in normally-cycling women, did not show a further increase with exercise. Basal testosterone levels were similar in normally-cycling and amenorrheic women however amenorrheic runners showed a greater exercise-induced increase in testosterone than did normally-cycling runners.

VII. APPENDIX B: ASSAY PROCEDURES

PIPETTING VARIABILITY

	1.0 ml	0.1 ml
	1.001	0.101
	0.998	0.101
	0.999	0.100
	0.998	0.099
	0.995	0.100
	0.999	0.099
	0.996	0.100
	0.998	0.099
	0.996	0.099
	0.995	0.100
	0.996	0.098
	1.000	0.099
	0.997	0.097
	0.994	0.100
	0.997	0.098
	0.994	0.101
	0.997	0.100
	0.996	0.100
	0.995	0.099
	0.995	0.099
Mean	0.997	0.100
S.D.	0.002	0.001
C.V.	0.2%	1.0%

ASSAY FOR THE DETERMINATION OF BLOOD LACTATE

REAGENTS

1. Ice cold 10% Perchloric Acid
2. β-Nicotinamide Adenine Dinucleotide (β-NAD)
3. Lactate Dehydrogenase Suspension (LDH)
4. Glycine Hydrazine Buffer - pH 9.2
5. Lactic Acid Standard Solution (0.4 mg/ml)
6. Lactic Acid Working Solution (0.6 ml lactic acid standard added to 2.4 ml water)

PREPARATION OF BLOOD

1. 1 ml samples of whole blood were transfered to test tubes containing 2 ml cold, 10% perchloric acid, vortexed, and left in an ice bath for at least 5 minutes.

2. Tubes were centrifuged at 2500 rpm for 10 minutes. The supernatant (protein-free filtrate) was drawn off into clean test tubes and frozen until time of assaying.

PROCEDURE

1. All standards and samples were assayed in triplicate.
2. Reagent was prepared in the following proportions:

    1.0 ml water

    0.5 ml glycine buffer

    0.025 ml LDH

    0.0025 g β-NAD

The quantity of reagent prepared depended on the number of samples to be assayed at one time. The above quantities were multiplied by $3(n + 6)$, where n = the number of samples to be assayed.

3. Standard curve points of 0, 12, 24, 36, 48, and 60 mg% were prepared containing respectively 0.00, 0.05, 0.10, 0.15, 0.20, and 0.25 ml of the lactic acid working solution. Reagent was added to bring the volume in each tube to 1.50 ml.

4. Sample tubes were prepared by adding 0.1 ml protein–free filtrate to 1.4 ml reagent.

5. All tubes were vortexed and left at room temperature for 45 minutes.

6. Standards and samples were read on the spectrophotometer against the blank (0%) at 340 nm.

7. The lactate concentrations were determined by applying sample O.D. readings to the linear regression of the standard data and multiplying by a correction factor of 0.6 (since 1.0 ml instead of 0.5 ml of blood was added to 2 ml perchloric acid).

LACTATE ASSAY VARIABILITY

	12mg%	24mg%	36mg%	48mg%	60mg%
	.171	.347	.518	.700	.860
	.193	.350	.518	.698	.850
	.176	.348	.512	.699	.873
	.174	.365	.530	.723	.868
	.175	.378	.550	.750	.925
	.172	.340	.515	.695	.855
	.190	.372	.515	.725	.905
	.174	.340	.540	.690	.865
Mean	.178	.359	.530	.710	.875
S.D	.008	.014	.017	.021	.026
C.V.	4.5%	3.9%	3.2%	3.0%	3.0%

RADIOIMMUNOASSAY FOR LUTEINIZING HORMONE

REAGENTS

1. LH I 125 - not more than 3uCi I 125 and 30 mg bovine serum albumin in 5.5 ml solution.
2. LH Antiserum (rabbit) - approximately 1 ul normal rabbit serum and 60 mg bovine serum albumin in 11 ml solution, anti-serum sufficient to bind at least 15% of 40 ng LH.
3. Second Antibody Reagent (Amerlex) (donkey) - anti-rabbit antibody coated on polymer particles of uniform diameter sufficient to bind at least 22 ug rabbit γ-globulin in approximately 110 ml solution, 3.0 ml normal human serum, EDTA.
4. Standard LH - 3.7, 9.2, 30, 92, 310 mIU/ml 2nd IRP-HMG and 50 mg bovine serum albumin in 1 ml solution.
5. Zero Standard - 250 mg bovine serum albumin in 5 ml solution.

PROCEDURE

1. 200 ul zero standard were pipetted into Non-Specific-Binding tubes.
2. 100 ul of the standards were pipetted into Standard tubes.
3. 100 ul of the samples were pipetted into Sample tubes.
4. 100 ul of LH antiserum were pipetted into all Standard and Sample tubes.
5. Tubes were vortexed, covered with plastic film, and incubated in a waterbath at body temperature for 1/2 hour.
6. 100 ul LH I 125 was added to each tube above and to Total Counts tubes.
7. Tubes were vortexed, covered with plastic film and incubated in a waterbath at room temperature for 1 hour.
8. 1.0 ml of second antibody reagent was added to all but the Total Counts tubes.
9. Tubes were vortexed and left for 10 minutes at room temperature .
10. Tubes were centrifuged at 1500g for 15 minutes.
11. Supernatant was drawn off using suction.

12. Tubes were counted in a gamma counter for 2 minutes.

LH ASSAY PERFORMANCE CHARACTERISTICS

i) Specificity - determined by cross-reactivity with the highly purified, structurally related human glycoprotein hormones.

Hormone - % Cross-reactivity

LH - 100.0

FSH - 2.4

TSH - 3.8

HCG - 19.0

ii) Precision/Reproducibility -

within assay variability - 3.5%

between assay variability - 6.6%

iii) Accuracy - recovery experiments resulted in a mean recovery of 104%.

iv) Sensitivity - the smallest amount of LH which could be distinguished from zero as defined by the 95% confidence limits of the within assay variation of the zero standard was 2.0 mIU/ml 2nd IRP-HMG.

RADIOIMMUNOASSAY FOR FOLLICLE-STIMULATING HORMONE

REAGENTS

1. FSH I 125 - not more than 3uCi I 125 and 30 mg bovine serum albumin in 5.5 ml solution.

2. FSH Antiserum (rabbit) - approximately 1 ul normal rabbit serum and 60 mg bovine serum albumin in 11 ml solution, anti-serum sufficient to bind at least 15% of 40 ng FSH.

3. Second Antibody Reagent (Amerlex) (donkey) - anti-rabbit antibody coated on polymer particles of uniform diameter sufficient to bind at least 22 ug rabbit γ-globulin in approximately 110 ml solution, 3.0 ml normal human serum, EDTA.

4. Standard FSH - 1.5, 5.5, 19, 61, 320 mIU/ml 2nd IRP-HMG and 100 mg bovine serum albumin in 2 ml solution.

5. Zero Standard - 250 mg bovine serum albumin in 5 ml solution.

PROCEDURE

1. 300 ul zero standard were pipetted into Non-Specific-Binding tubes.

2. 200 ul of the standards were pipetted into Standard tubes.

3. 200 ul of the samples were pipetted into Sample tubes.

4. 100 ul of FSH antiserum were pipetted into all Standard and Sample tubes.

5. Tubes were vortexed, covered with plastic film, and incubated in a waterbath at body temperature for 1/2 hour.

6. 100 ul FSH I 125 was added to each tube above and to Total Counts tubes.

7. Tubes were vortexed, covered with plastic film and incubated in a waterbath at body temperature for 1 hour.

8. 1.0 ml of second antibody reagent was added to all but the Total Counts tubes.

9.Tubes were vortexed and left for 10 minutes at room temperature .

10. Tubes were centrifuged at 1500g for 15 minutes.

11. Supernatant was drawn off using suction.

12. Tubes were counted in a gamma counter for 2 minutes.

FSH ASSAY PERFORMANCE CHARACTERISTICS

i) Specificity - determined by cross-reactivity with the highly purified, structurally related human glycoprotein hormones.

Hormone - % Cross-reactivity

FSH - 100.0

TSH - 0.35

LH - 0.10

HCG - <0.03

ii) Precision/Reproducibility -

within assay variability - 4.8%

between assay variability - 9.2%

iii) Accuracy - recovery experiments resulted in a mean recovery of 104%.

iv) Sensitivity - the smallest amount of FSH which could be distinguished from zero as defined by the 95% confidence limits of the within assay variation of the zero standard was 0.8 mIU/ml 2nd IRP-HMG.

ASSESSMENT OF BODY COMPOSITION

Four pieces of data were required for the determination of percent body fat;

i) Water density was determined from water temperature.

ii) The subject's dry weight.

iii) The subject's weight in water was determined with the subject totally submerged in water, holding a maximum inspiration, and seated on a weighing chair suspended from a tensiometer. A minimum of five recordings were taken and the lowest recorded value was used.

iv) The subject's vital capacity was measured with the subject seated on the weighing chair in the water tank with water at neck level. A minimum of five readings were taken and the highest value was used. Residual volume was estimated as 30% of the vital capacity.

VIII. APPENDIX C: RAW DATA

TRAINING PROGRESSION SHOWING NUMBER OF PLATES LIFTED FOR EACH EXERCISE EACH WEEK AND DURING THE PRE-TEST AND POST-TEST

SUBJECT A:

EXERCISE	PRE	WEEK 1	2	3	4	5	6	7	8	POST
Pullover	3	3	3	4	4	4	4	4	5	5
Arm Cross	2	2	2	2	2	3	3	3	3	3
Decline Press	2	3	3	4	4	4	5	4	4	4
Hip & Back	6	6	7	8	8	9	9	8	8	8
Quadriceps	6	7	8	9	9	10	11	11	12	12
Leg Curls	5	4	5	5	5	6	6	5	6	6

SUBJECT B:

EXERCISE	PRE	WEEK 1	2	3	4	5	6	7	8	POST
Pullover	3	4	4	4	4	5	5	5	5	5
Arm Cross	2	2	2	3	3	3	3	3	3	3
Decline Press	2	3	3	3	4	4	4	5	5	5
Hip & Back	6	7	8	8	8	8	7	7	7	8
Quadriceps	5	6	7	10	12	13	14	15	15	15
Leg Curls	4	3	4	5	6	6	6	6	6	6

TRAINING PROGRESSION SHOWING NUMBER OF PLATES LIFTED FOR EACH EXERCISE EACH WEEK AND DURING THE PRE-TEST AND POST-TEST

SUBJECT C: WEEK

EXERCISE	PRE	1	2	3	4	5	6	7	8	POST
Pullover	2		3			4		4		4
Arm Cross	2		2			3		3		3
Decline Press	2		2			3		4		4
Hip & Back	5		6			7		7		7
Quadriceps	6		6			8		9		10
Leg Curls	4		4			5		5		5

SUBJECT E: WEEK

EXERCISE	PRE	1	2	3	4	5	6	7	8	POST
Pullover	3	3	3	3	4	4	4	4	4	4
Arm Cross	2	2	2	2	2	2	2	2	2	2
Decline Press	2	2	2	2	2	3	3	3	3	3
Hip & Back	5	5	6	6	7	7	7	7	7	7
Quadriceps	6	6	7	7	8	9	10	12	12	13
Leg Curls	3	3	3	3	4	4	4	5	5	4

TRAINING PROGRESSION SHOWING NUMBER OF PLATES LIFTED FOR EACH EXERCISE EACH WEEK AND DURING THE PRE-TEST AND POST-TEST

SUBJECT F:

	WEEK									
EXERCISE	PRE	1	2	3	4	5	6	7	8	POST
Pullover	4	4	5	5	5	5	5	5	5	5
Arm Cross	2	2	2	3	4	4	4	4	4	4
Decline Press	3	3	3	4	4	4	4	4	5	5
Hip & Back	6	8	8	9	9	9	9	9	9	9
Quadriceps	7	13	17	19	20	22	23	24	24	24
Leg Curls	5	5	5	6	6	6	7	7	7	6

SUBJECT H:

	WEEK									
EXERCISE	PRE	1	2	3	4	5	6	7	8	POST
Pullover	2		4	4	4	4				5
Arm Cross	2		2	3	3	3				4
Decline Press	2		2	3	3	4				5
Hip & Back	5		6	6	6	7				7
Quadriceps	6		8	8	9	9				10
Leg Curls	3		4	4	5	5				5

TRAINING PROGRESSION SHOWING NUMBER OF PLATES LIFTED FOR EACH EXERCISE EACH WEEK AND DURING THE PRE-TEST AND POST-TEST

SUBJECT I: WEEK

EXERCISE	PRE	1	2	3	4	5	6	7	8	POST
Pullover	4	4	4	4	4	5	5	5	5	5
Arm Cross	2	2	2	2	2	2	3	3	3	3
Decline Press	2	2	2	2	2	2	3	3	3	3
Hip & Back	6	6	6	7	7	8	8	8	8	9
Quadriceps	6	6	6	7	7	8	8	8	8	9
Leg Curls	4	4	4	5	5	5	5	5	5	5

BODY COMPOSITION BEFORE AND AFTER TRAINING

Before Training

Subject	Weight (kg)	Percent Fat	Kilograms Fat	Kilograms Lean
A	56.4	23.9	13.5	42.9
B	63.6	31.4	20.0	43.6
C	59.1	20.3	12.0	47.1
E	47.3	17.8	8.4	38.9
F	60.9	25.1	14.5	45.6
H	55.9	25.9	14.5	41.4
I	63.2	25.2	15.9	47.3

After Training

A	56.8	24.7	14.0	42.8
B	66.4	32.9	21.8	44.6
C	60.0	19.6	11.8	48.2
E	45.9	21.2	9.7	36.2
F	61.8	25.1	15.5	46.3
I	64.4	22.5	14.5	50.1

HEMATOCRIT (% RBC BY VOLUME)

Pre-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	38.0	38.0	38.0	39.0	41.0	40.0	40.0	40.0	39.0	37.0	37.0
B	40.0	40.0	43.0	41.5	43.5	–	45.0	–	46.5	40.0	43.0
C	42.5	41.0	37.5	39.0	41.0	43.0	42.0	44.0	43.0	41.0	40.0
E	40.0	41.5	42.0	43.0	42.0	42.0	42.0	41.0	40.0	38.5	39.0
F	41.0	39.0	39.0	44.0	46.0	44.0	45.0	45.0	43.0	40.0	38.0
H	32.0	38.0	–	–	43.0	42.0	41.0	42.0	41.0	36.0	38.0
I	38.5	37.0	37.0	42.0	42.0	42.0	40.0	42.0	40.0	39.0	38.0

Post-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	37.5	37.0	36.5	40.5	41.0	39.0	40.0	40.0	40.5	39.0	38.5
B	40.5	39.0	40.5	44.0	–	42.5	43.0	44.0	–	44.0	40.5
C	44.5	44.5	44.0	44.0	44.0	44.0	44.0	45.0	44.0	42.0	43.5
E	40.5	39.0	39.5	41.0	42.0	40.5	42.5	–	44.0	40.0	38.0
F	41.0	41.0	39.5	42.5	43.0	42.5	44.0	46.0	45.0	41.5	40.0
H	38.0	36.0	44.0	43.0	43.0	43.0	42.0	44.0	42.0	39.0	39.0
I	37.5	36.0	35.5	39.0	41.0	40.0	–	39.0	39.0	36.0	–

HEMATOCRIT (% OF BASELINE)

Pre-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	100.0	100.0	100.0	102.6	107.9	105.3	105.3	105.3	102.6	97.4	97.4
B	100.0	100.0	107.5	103.8	108.8	–	112.5	–	116.3	100.0	107.5
C	100.0	100.0	89.3	92.9	97.6	102.4	100.0	104.8	102.4	97.6	95.2
E	100.0	100.0	102.4	107.5	102.4	102.4	102.4	100.0	97.6	93.9	95.1
F	100.0	100.0	97.5	110.0	115.0	110.0	112.5	112.5	107.5	100.0	95.0
H	100.0	100.0	–	–	112.9	120.0	117.1	120.0	117.1	102.9	108.6
I	100.0	100.0	97.4	110.5	110.5	110.5	105.3	110.5	105.3	102.6	100.0

Post-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	100.0	100.0	98.7	109.5	110.8	105.4	108.1	108.1	109.5	105.4	104.1
B	100.0	100.0	101.3	110.0	–	106.3	107.5	110.0	–	110.0	101.3
C	100.0	100.0	98.9	98.9	98.9	98.9	98.9	101.1	98.9	94.4	97.8
E	100.0	100.0	98.8	102.5	105.0	101.3	106.3	–	110.0	100.0	95.0
F	100.0	100.0	96.3	103.7	104.9	103.7	107.3	112.2	109.8	101.2	97.6
H	100.0	100.0	118.9	116.2	116.2	116.2	113.5	118.9	113.5	105.4	105.4
I	100.0	100.0	95.9	105.4	110.8	108.1	–	105.4	105.4	97.3	–

BLOOD LACTATE (mg%)

Pre-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	4.37	4.37	4.91	18.36	18.56	23.11	20.88	20.96	32.59	15.39	9.78
B	2.98	4.28	4.01	14.47	15.47	33.90	–	46.82	–	36.17	19.22
C	5.62	–	–	–	27.31	31.85	42.90	48.01	38.22	28.34	21.74
E	5.09	5.55	5.55	33.58	37.53	38.92	44.27	40.98	48.80	32.75	19.79
F	5.59	5.00	5.29	17.35	33.07	26.62	35.27	43.67	44.01	31.12	22.59
H	2.98	3.47	3.21	–	22.62	24.20	–	55.92	30.64	20.24	23.87
I	5.92	5.71	5.42	18.06	46.01	22.87	30.28	47.15	37.18	19.87	14.27

Post-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	2.72	3.02	2.64	13.33	19.18	14.58	23.29	40.43	39.77	26.29	19.84
B	3.18	3.02	3.11	39.61	28.96	23.87	32.99	66.26	37.19	18.86	–
C	3.26	2.52	2.76	17.87	17.87	19.99	43.75	53.80	67.68	42.53	31.92
E	4.10	3.94	3.35	29.62	28.12	33.96	56.39	71.02	63.07	47.53	29.78
F	5.84	4.60	5.17	17.06	22.04	28.36	16.07	83.32	85.80	65.88	37.82
H	4.78	3.18	3.94	25.85	21.17	36.73	37.14	59.88	64.33	34.38	23.26
I	6.29	5.40	5.15	34.33	38.48	–	–	59.08	49.69	32.59	18.56

LH (mIU/ml)

Pre-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	9.27	8.16	7.93	8.11	7.16	4.53	7.48	8.17	7.11	7.80	5.94
B	7.92	6.72	6.68	16.17	16.53	12.71	14.25	18.53	18.05	17.31	14.81
C	4.10	6.23	6.54	6.65	4.85	–	–	4.84	3.74	3.63	7.23
E	7.52	6.79	6.27	14.96	12.24	11.35	10.03	10.23	9.83	9.64	7.50
F	7.99	8.15	7.69	8.21	8.65	6.63	6.86	7.16	7.95	6.53	6.83
H	15.64	14.25	11.47	–	23.29	20.71	13.58	17.65	14.91	14.61	12.50
I	6.58	4.83	5.36	7.49	8.23	5.46	4.13	7.37	7.48	4.73	4.25

Post-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	6.30	6.69	5.53	4.75	13.15	11.58	11.44	13.12	12.39	9.14	8.30
B	9.63	9.55	11.73	11.76	14.08	12.88	13.64	14.20	–	13.67	11.05
C	4.29	3.15	2.51	3.17	8.52	7.37	8.28	7.85	6.77	5.28	5.01
E	18.30	15.58	13.73	12.83	12.60	10.95	12.26	12.68	13.03	10.86	9.87
F	6.03	9.08	7.66	6.87	8.71	–	10.50	10.51	9.51	7.03	6.42
H	7.24	6.18	10.55	12.51	10.81	11.06	10.19	17.47	13.28	10.88	9.10
I	3.88	3.76	3.18	5.41	7.04	6.66	–	3.34	5.76	4.20	4.65

LH (% OF BASELINE)

Pre-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	100.0	100.0	91.0	93.1	82.2	52.0	85.8	93.7	81.6	89.5	68.2
B	100.0	100.0	91.3	220.9	225.8	173.6	194.7	253.1	246.6	236.5	202.3
C	100.0	100.0	126.6	128.8	93.9	–	–	93.71	72.4	70.3	140.0
E	100.0	100.0	87.6	209.1	171.1	158.6	140.2	143.0	137.4	134.7	104.8
F	100.0	100.0	95.3	101.7	107.2	82.2	85.0	88.7	98.5	80.9	84.6
H	100.0	100.0	76.7	–	155.8	138.6	191.0	118.1	100.0	97.8	83.6
I	100.0	100.0	94.0	131.3	144.3	95.7	72.4	129.2	131.1	82.9	74.5

Post-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	100.0	100.0	85.1	73.1	202.5	178.3	176.1	202.0	190.8	140.7	127.8
B	100.0	100.0	122.3	122.6	146.8	134.3	142.2	148.1	–	142.8	115.2
C	100.0	100.0	67.5	85.2	229.0	198.1	222.6	211.0	182.0	141.9	134.7
E	100.0	100.0	81.1	75.7	74.4	64.6	72.4	74.9	76.9	64.1	58.3
F	100.0	100.0	101.4	90.9	115.3	–	139.0	139.1	125.9	93.1	95.0
H	100.0	100.0	157.2	186.4	161.1	164.8	151.9	260.4	197.9	167.1	135.6
I	100.0	100.0	83.2	141.6	184.3	172.8	–	87.4	150.8	109.9	121.7

FSH (mIU/ml)

Pre-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	11.67	10.31	11.08	9.70	–	9.37	8.84	10.89	10.40	9.92	8.97
B	7.56	6.92	7.24	8.51	9.79	8.48	6.16	9.32	8.34>	8.17	9.29
C	8.14	8.67	7.67	7.11	7.72	5.31	–	7.24	5.81	6.21	7.09
E	9.13	8.51	8.69	9.88	9.22	9.51	7.41	8.65	8.05	8.08	7.96
F	7.15	6.79	6.89	7.19	8.59	6.79	7.58	7.86	7.07	6.63	6.56
H	7.04	6.38	5.56	–	10.03	9.04	6.77	9.21	7.51	7.77	8.19
I	4.96	4.82	4.58	6.63	5.35	5.53	4.83	5.37	5.36	4.95	4.34

Post-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	4.32	10.78	10.52	9.01	12.69	11.12	–	12.91	12.73	12.03	12.38
B	9.05	7.91	8.90	9.04	10.12	9.36	9.82	–	–	8.76	9.23
C	6.05	5.86	5.43	5.87	2.89	5.67	6.46	–	6.43	5.27	6.50
E	–	6.11	6.54	6.23	6.01	6.10	5.18	5.03	5.04	4.29	4.08
F	4.44	7.35	7.03	6.98	7.66	4.52	7.24	6.55	7.79	5.90	–
H	9.11	7.59	10.37	12.30	10.94	11.16	10.46	12.94	11.79	9.68	10.07
I	–	5.31	5.60	5.53	5.85	5.93	–	3.85	5.18	4.67	4.08

FSH (% OF BASELINE)

Pre-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	100.0	100.0	100.8	88.3	–	85.3	84.0	99.1	94.6	90.3	81.6
B	100.0	100.0	100.0	117.5	135.2	117.1	–	128.7	115.2	112.8	128.3
C	100.0	100.0	91.3	84.6	91.9	63.2	–	86.1	69.1	73.9	84.4
E	100.0	100.0	98.5	112.0	104.5	107.8	84.0	98.1	91.3	91.6	90.2
F	100.0	100.0	98.9	103.2	123.2	97.4	108.8	112.8	101.4	95.1	94.1
H	100.0	100.0	82.9	–	149.5	134.7	100.9	137.3	111.9	115.8	122.1
I	100.0	100.0	93.7	135.6	109.4	113.1	98.8	109.8	101.2	101.2	88.3

Post-Test Samples

Subject	1	2	3	4	5	6	7	8	9	10	11
A	100.0	100.0	139.3	119.3	168.1	147.3	–	171.0	168.6	159.3	164.0
B	100.0	100.0	105.0	106.6	119.3	110.4	–	–	–	103.3	108.8
C	100.0	100.0	91.2	98.6	–	95.2	108.5	–	108.0	88.5	109.2
E	100.0	100.0	107.0	102.0	98.4	99.8	84.8	82.3	82.5	70.2	66.8
F	100.0	100.0	119.3	118.4	129.9	76.7	122.8	111.1	132.1	100.1	–
H	100.0	100.0	123.0	147.3	131.0	133.7	125.3	155.0	141.2	115.9	121.0
I	100.0	100.0	105.5	104.1	110.2	111.7	–	72.5	97.6	87.9	76.8

IX. APPENDIX D: STATISTICAL PROCEDURES

SUMMARY OF PAIRED T-TEST FOR TO DETERMINE TRAINING EFFECTS

PULLOVER	Pre	Post
Mean	3.00	4.70
S.E.M.	0.31	0.18
D.F.	6	
t-value	5.99*	

ARM CROSS	Pre	Post
Mean	2.00	3.14
S.E.M.	0.00	0.26
D.F.	6	
t-value	4.38*	

DECLINE PRESS	Pre	Post
Mean	2.14	4.14
S.E.M.	0.14	0.34
D.F.	6	
t-value	6.48*	

* $p<0.01$

HIP AND BACK	Pre	Post
Mean	5.57	7.71
S.E.M.	0.20	0.29
D.F.	6	
t-value	15.00*	

QUADRICEPS	Pre	Post
Mean	6.00	13.29
S.E.M.	0.22	1.90
D.F.	6	
t-value	3.94*	

LEG CURLS	Pre	Post
Mean	4.00	5.29
S.E.M.	0.31	0.29
D.F.	6	
t-value	6.97*	

* $p<0.01$

SUMMARY OF PAIRED T-TEST FOR BODY COMPOSITION

	Pre	Post
Mean	23.95	24.33
S.E.M.	1.91	1.91
D.F.	5	
t-value	0.45*	

* $p>0.05$

SUMMARY OF 2-WAY ANOVA FOR LACTATE

Source of Variation	Sum of Squares	Degrees of Freedom	Mean Square	F Ratio	Tail Probability
Training	1157.32	1	1157.32	4.03	0.068
Time	39599.16	10	3959.92	72.99	0.0
Interaction	2354.94	10	235.49	4.34	0.0
Error	6510.42	120	54.25		

SUMMARY OF TUKEY TEST FOR LACTATE.

		Pre–test samples											Post–test samples									
		1	2	3	4	5	6	7	8	9	10	11	1	2	3	4	5	6	7	8	9	10	11
	1		–	–	*	*	*	*	*	*	*	*	–	–	–	*	*	*	*	*	*	*	*
	2			–	*	*	*	*	*	*	*	*	–	–	–	*	*	*	*	*	*	*	*
	3				*	*	*	*	*	*	*	*	–	–	–	*	*	*	*	*	*	*	*
	4					*	*	*	*	*	–	–	*	*	*	–	–	–	*	*	*	*	–
	5						–	–	*	*	–	*	*	*	*	–	–	–	*	*	*	*	–
Pre	6							–	*	*	–	*	*	*	*	–	–	–	*	*	*	*	–
test	7								*	–	*	*	*	*	*	*	*	*	*	*	*	–	*
	8									–	*	*	*	*	*	*	*	*	–	*	*	–	*
	9										*	*	*	*	*	*	*	*	–	*	*	–	*
	10											–	*	*	*	–	–	–	*	*	*	*	–
	11												*	*	*	–	–	–	*	*	*	*	–
	1													–	–	*	*	*	*	*	*	*	*
	2														–	*	*	*	*	*	*	*	*
	3															*	*	*	*	*	*	*	*
	4																–	–	*	*	*	*	–
	5																	–	*	*	*	*	–
Post	6																		*	*	*	*	–
test	7																			*	*	–	*
	8																				–	*	*
	9																					*	*
	10																						*
	11																						

* critical difference between means > 7.78 significant at $p<0.05$.

SUMMARY OF 2-WAY ANOVA FOR LH

Source of Variation	Sum of Squares	Degrees of Freedom	Mean Square	F Ratio	Tail Probability
Training	0.31	1	0.31	0.0	0.96
Time	212.10	10	21.21	3.81	0.0
Interaction	34.54	10	3.45	0.62	0.80
Error	668.81	120	5.57		

SUMMARY OF TUKEY TEST FOR LUTEINIZING HORMONE

		Pre–Test Samples											Post–Test Samples									
		1	2	3	4	5	6	7	8	9	10	11	1	2	3	4	5	6	7	8	9	10	11
Pre test	1		–	–	–	*	–	–	–	–	–	–	–	–	–	–	–	–	*	*	–	–	–
	2			–	–	*	–	–	*	–	–	–	–	–	–	–	*	–	*	*	–	–	–
	3				*	*	–	–	*	–	–	–	–	–	–	–	*	–	*	*	*	–	–
	4					–	–	–	–	–	–	–	–	*	–	–	–	–	–	–	–	–	*
	5						*	–	–	–	–	*	*	*	*	*	–	–	–	–	–	*	*
	6							–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
	7								–	–	–	–	–	–	–	–	–	–	–	–	–	–	*
	8									–	–	–	*	*	*	–	–	–	–	–	–	–	–
	9										–	–	–	–	–	–	–	–	–	–	–	–	–
	10											–	–	–	–	–	–	–	–	–	–	–	–
	11												–	–	–	–	–	–	*	*	–	–	–
Post test	1													–	–	–	*	–	*	*	–	–	–
	2														–	–	*	–	*	*	–	–	–
	3															–	*	–	*	*	–	–	–
	4																*	–	*	*	–	–	–
	5																	–	–	–	–	–	*
	6																		–	–	–	–	–
	7																			–	–	*	*
	8																				–	–	–
	9																					–	–
	10																						–
	11																						

* critical difference between means > 2.49 significant at $p<0.05$.

SUMMARY OF 2-WAY ANOVA FOR FSH

Source of Variation	Sum of Squares	Degrees of Freedom	Mean Square	F Ratio	Tail Probability
Training	0.02	1	0.02	0.0	0.98
Time	18.66	10	1.87	1.25	0.27
Interaction	11.80	10	1.18	0.79	0.64
Error	178.91	120	1.49		

X. APPENDIX E: ADDITIONAL INFORMATION

CONSENT FORM

I, _____, agree to participate in a study on hormonal changes with exercise training, as conducted by Meredith Galbraith under the supervision of Dr. A.N. Belcastro, Department of Physical Education, and Dr. D.C. Cumming, Department of Obstetrics and Gynecology. The nature of the study, my role, and the experimental procedures for which I will be subject have all been fully explained to me.

I understand that:

1) I will be involved in an 8 week training period involving 3 half-hour training periods per week.
2) My percentage body fat will be measured using the underwater weighing technique, both before and after the training program.
3) I will be studied over single exercise periods, before and after the actual training program, where an intravenous catheter will be placed in my arm for the removal of small serial blood samples for blood hormone level analysis.
4) That once I have agreed to participate in the study I may still withdraw at any time.

Date _____ Signature _____

Witness _____

QUESTIONNAIRE

NAME:

ADDRESS: PHONE NO.:

EDUCATION:

OCCUPATION:

AGE: DATE OF BIRTH:

GENERAL HEALTH:

1) How old were you when you first started menstruating?

2) How long have you been menstruating?

3) Do you consider your periods to be regular?

4) How many days are there usually between your periods?

5) How many days do your periods usually last?

6) Do you consider your flow to be light? medium? heavy?

7) Do you experience premenstrual discomfort?
If yes, what sort, to what degree, and for how long?

8) Do you experience menstrual discomfort?
If yes, what sort, to what degree, and for how long?

9) Have you had any pregnancies, miscarriages, or abortions?

10) Have your periods ever stopped?

If yes i) Did they stop suddenly with no warning?
Did they gradually taper off?

ii) Did you deliberately attempt to change your weight before your periods stopped?

iii) Was loss of menses associated with a change in body weight?

iv) Do you associate your periods stopping with a change in exercise?
If so, how long before or after you began exercising?

v) Do you associate your periods stopping with any change in stress, diet, medication, environment, illness or other factor?

11) Do you associate any changes in your menses with changes in body weight?

12) Do you associate any changes in your menses with changes in exercise?

13) Do you associate any changes in your menses with a change in stress, diet, medication, environment, illness or other factor?

14) How much did you weigh when you first started menstruating?

15) How much do you weigh now?

16) How tall are you?

17) What type of frame do you have?

18) What is the most you have ever weighed in your adult life?

19) What is the least you have ever weighed in your adult life?

20) Briefly describe any major weight changes.

21) Do you follow any special diet?

22) Do you restrict your caloric intake?
If yes, to what amount?

23) Have your eating habits changed significantly at any time in recent years?

24) Do you think that your eating habits alter when you are involved in regular exercise?

25) Do you exercise regularly?
If yes, give details.

26) Do you use oral contraceptives?
If yes, what type, what dose?

PMTQ

NAME _____ AGE ____

Marital Status M / S / W / D
Contraception None IUD Pill Other
Occupation _____

DURING THE WEEK FOLLOWING THE END OF YOUR PERIOD AND DURING THE WEEK BEFORE YOUR PERIOD, DO YOU EXPERIENCE ANY OF THESE SYMPTOMS? CHECK ONE BOX FOR EACH SYMPTOM BOTH DURING THE WEEK FOLLOWING YOUR PERIOD AND THE WEEK PRECEDING YOUR PERIOD.

SYMPTOMS	WEEK FOLLOWING PERIOD				WEEK BEFORE PERIOD			
	None	Mild	Mod	Severe	None	Mild	Mod	Severe
Abdominal Bloating								
Anxiety								
Breast Tenderness								
Confusion								
Craving for Sweets								
Crying								
Depression								
Dizziness or Fainting								
Fatigue								
Forgetfulness								
Headache								
Heart Pounding								
Heart Pounding								
Increased Appetite								
Insomnia								
Irritability								
Mood Changes								
Nervous Tension								
Swelling of Hands/feet								
Weight Gain								

FOR THE BEGINNER

1. INTRODUCTION

The purpose of this programme is to increase an individual's general overall body strength and to outline some important principles that should be adhered to if maximum benefits are to be achieved. If the beginner executes this programme properly, a significant increase in strength and a moderate increase in muscle mass will be obtained in the majority of individuals. Weight training will not significantly improve the functioning of the cardiovascular system (heart and blood vessels), general body coordination or cause an automatic loss of weight.

2. PRINCIPLES OF STRENGTH TRAINING

(1) Overload:

The overload principle is the most important principle of weight training. A muscle will grow bigger and stronger only when the muscle has imposed upon it a demand which is not easily met! In fact, best results are obtained when a muscle group is worked to the POINT OF MOMENTARY FAILURE.

The following is an example of the application of the overload principle: an exercise requires a set of eight to ten repetitions. The trainee selects a resistance that will cause him to fail somewhere between eight to ten repetitions. He performs as many full repetitions as possible, but he does not stop there. He should attempt as many partial repetitions as possible; he continues until he is UNABLE TO MOVE THE RESISTANCE AT ALL!! Anything less than maximal all-out effort falls short of maximum

training stimulus, and poor results will be obtained.

One set of any exercise performed as recommended above is far more productive for results than any number of sets which are not performed to the point of failure.

Working to the point of failure is not a dangerous practice in strength training. On the contrary, the last repetition or partial repetition before failure is actually the safest repetition from the standpoint of injury to muscle and joints. Less tension and force are being produced by a muscle as it begins to fail; therefore, less stress is placed upon muscle structure and joint structure.

It is true that some results can be obtained from less than maximum effort, but BEST results come from maximum effort, and because maximum effort is also SAFE, it is highly recommended.

The overload principle has been explained in detail because it is frequently misunderstood and in many cases is not applied. A weight training programme often degenerates into a boring, lengthy form of manual labour, without significant results. Hard work in strength training refers to INTENSE EFFORT, not a greater "amount" of work (more sets, exercises, workouts).

(2) Heavy Resistance:

When the overload principle is applied to working a muscle, the response of the muscle will depend upon the amount of resistance used in the work. If the resistance is light so that failure occurs only after a relatively high number of repetitions have been performed, the training effect will be primarily an increase in muscular endurance. However, if the resistance is heavy so

that failure occurs after a relatively low number of repetitions have been performed, the training effect will be primarily an increase in muscular strength.

(3) Repetitions:

What is the optimum number of contractions per hour (repetitions)? The answer to this question has not been answered clearly on a scientific basis and generally is arbitrary depending on the goal of the programme. However, the following observations have been made.

Extremely low repetitions (1-3) are very effective for building strength. Using very low repetitions is an advanced form of training. Because of the heavy poundages that are used in conjunction with extremely low repetitions, there is the possibility of injury in the case of the inexperienced trainees. Recovery ability is reduced markedly when very low repetitions are utilized. Even very advanced competitive lifters can train to maximum using very low repetitions only once per week (sometimes once per month). Less muscle size and local muscular endurance are developed when extremely low repetitions are employed. On the other hand, there is a large range of repetitions (5-20) which is effective in producing strength, muscle size and local muscular endurance and which does not unduly tax the recovery ability of the muscle group. This is the range of repetitions that will be used in this strength training programme.

(4) Sets:

The term "set" refers to a bout of repetitions. "Two sets, eight repetitions" of an exercise means two bouts of executions

of an exercise movement, with a short rest between the two bouts.

Two to three sets per exercise is optimum. Research has shown that performing more than three sets does not significantly improve results. On the contrary, more than three sets will often hinder the recovery ability of the muscle and will slow or stop results altogether.

Most exercises then, based on reported research studies, will require two to three sets of between five to twenty repetitions.

(5) Progressive Resistance:

In order to adhere to the overload principle and the heavy resistance principle, it is necessary to gradually increase the exercise resistance as the muscle becomes stronger.

An example: An exercise requires two sets of eight to ten repetitions. In a period of time, nine and finally ten repetitions will be possible with that resistance since the muscle has increased in strength. When ten repetitions are possible on either of the two sets, five to ten pounds is added to the resistance. The resistance is progressively increased so that failure will always occur between eight to ten repetitions.

(6) Specificity:

Gains in strength are largely specific to the movement patterns exercised. Therefore, since this programme is aimed at increasing general body strength, a programme which effects all the major muscle groups will be emphasized.

(7) Frequency:

Trainees who are practising overload and heavy, progressive resistance training cannot, in the vast majority of cases, tolerate

daily training. In fact, daily training of this nature will usually result in losses of strength and muscle size in most individuals. The actual training effect (increases size and strength) occurs during the REST PERIOD following the exercise. As a general rule, at least one full day of rest from resistance training is required to allow for recovery and to allow the training effect to occur. Three training sessions per week (Monday, Wednesday, Friday, or Tuesday, Thursday, Saturday) is the usual practice. This is probably a reasonable procedure for most persons. The extra day of rest at the end of each week is important for good results.

For some trainees, three workouts per week is too much because of various levels of initial fitness or living habits. Two workouts per week (Monday, Thursday or Tuesday, Friday) can be very effective. This is particularly true of individuals who are engaged in vigorous activity or sport, or who have jobs involving hard manual labour, besides their weight training programme. These people may consider a twice weekly programme which will also produce significant results.

OVER-TRAINING, or STALENESS, which is often responsible for poor progress, is characterized by too many workouts per week and workouts which are too long (too many exercises and sets).

UNDER-TRAINING, which may be responsible for poor results, is almost always characterized by the absence of the overload principle (working each set to the absolute point of failure). Under-training rarely results from too few workouts, sets or exercises.

3. RATE OF PROGRESS

The rate of progress will vary considerably among trainees; however, all trainees in good health who are training properly will make progress. In the first month, the improvements in strength performance are due mainly to increases in skill in executing the exercises. In many months that follow, gains in strength will be associated with increases in muscle size and a general increase in body weight. Best progress in strength training results from regular training over many months and years.

The following six factors may influence gains in strength and are briefly reviewed.

1. Body Type:

Weight trainees who tend to fall into the mesomorphic somatype group seem to get better results from weight lifting than individuals who are wither ectomorphic (predominantly slim) or endomorphic (predominantly fat) in body build.

2. Muscle Group Trained:

The upper body will respond better to resistance training than the musculature of the lower body because the upper body muscles are innervated by a greater number of nerves. This allows the upper body musculature to produce a greater firing rate of muscle fibres and consequently will allow the muscle to exert more force.

Secondly, it is difficult to exercise the lower body muscles to complete failure because the knee extensions will fail long before the extensors of the hip are fully fatigued. Consequently, the hip extensors will not receive a full training effect.

3. Age:

Older individuals who have been shown to have less nerve and muscle cells, will not have as great a training effect as those persons who are in early adulthood.

4. State of Prior Training:

Persons with no prior weight training experience will show rapid improvement in the early part of their resistance training programme and will gradually level off as they progress. Since some muscle groups are in a higher state of training than others, when a weight training programme is initiated, these particular muscle group will respond less. As a general rule, the closer a weight trainee gets to his/her maximum strength level, the higher the stimulus needed to fire the high threshold motor neurons required to overcome the heavy resistance levels which are loaded on the muscle.

5. Sex:

Due to the presence of the hormone testosterone in males, they will respond to weight training better than females. However, women can develop their lower body as much as males in relative strength.

6. Recovery Ability:

As previously mentioned, as one gets closer to one's maximum strength level, higher nerve stimulus is required to facilitate muscular contraction. Pushing oneself to his physiologic limit requires more time for the body to recover. Consequently, beginners can train three times a week and recover on the alternate or "off days". However, more experienced lifters will find

that they may only be capable of lifting two times a week because they will take longer to fully recover.

If the trainee appears to be making little or no progress after two months of training, check the following points:

(1) Are you using the overload principle?

(2) Are you overtraining (too many workouts and sets; you may have to cut down to two workouts per week)? DON'T TRAIN MORE IN FRUSTRATION.

(3) Are you getting enough rest!? Eight hours sleep per night regularly is the minimum requirement.

(4) Are you eating well? "Well" refers to quality and quantity of food.

4. QUESTIONS??

Like many forms of training programmes, the beginner often has many questions regarding specific aspects of the programme. This is particularly true of weight training in which a number of "old wives tales" have been perpetuated among experienced lifters or by individuals totally unaware what takes place in a weight training facility. With this in mind, the following section will try and provide answers to the more common questions which the beginner may have and hopefully dispel any doubts or fears a beginner may have encountered.

1. Who should not lift weights?

Persons who are sick, especially if they have some type of circulatory disorder, should not exercise with weights unless they have permission by their physician. In terms of athletes, there is no harm done by the athlete who engages in weight lifting during the season of his/her sports participation.

2. How much can one improve physique through weight training?

Improvement in physique through weight training occurs in three ways: muscle girths are increased, fatty tissue is reduced, and posture is improved. The degree to which this will occur depends largely on the factors mentioned in the "Rate of Progress" section.

3. How long must one train before results take place?

The rapidity of increase in muscle siz, strength, and other related factors depends upon body type, intensity of training, living habits, etc. However, if the principles outlined in this paper are properly followed, definite changes in muscle size can be noted within four (4) weeks after training starts.

4. Will weight lifting cause "muscle-boundness"?

"Muscle-boundness" is a term used to describe heavily muscled individuals who are slow and awkward in their movements. The results of research studies to date have not provided a conclusive answer, but the evidence indicates: (1) muscle-boundness is a figment of the imagination and no such condition usually exists, (2) persons who have exercised with weights and/or are muscularly strong can move faster than the average person, and are just as well coordinated, (3) weight lifters have just as much and perhaps more bodily flexibility than does the average person. In fact, if properly done, resistance training can actually increase flexibility!

5. Do persons who have weight training get fat when they discontinue training?

There is no reason whatsoever to believe that weight lifting per se is conducive to obesity either during training or upon cessation. Upon discontinuance of training, one needs only to be careful of his/her diet to avoid obesity. This is due to the fact that during heavy training, an individual gets into the habit of eating more and with the cessation of training often does not reduce their food intake to coincide with the reduction in energy expenditure.

6. How dangerous is weight lifting?

Weight lifting is a very safe activity. In fact, if proper safety precautions, such as spotters, are observed, injury is practically impossible.

7. What about dietary supplement or "super-foods"?

Unless a person is definitely deficient in some food element, any extra amount of the substance taken in generally is passed off. Tablets, unique diets, and special "formulae" can best be passed up and the money saved. The best "super-food" is a good balanced diet!

5. ADVANCED TRAINING

a) Nautilus Strength Training Regime

In many barbell and barbell-like exercises, there is no available resistance at either end of the movement. In a conventional bench press exercise, for example, there is no resistance at the top of the movement since the trainee is "locked out" and supports the weight without the requirement for muscular involvement.

Barbell exercises are also limited by the fact that the resistance remains more or less constant during the entire range of movement; which means that you will always be limited to resistance that you can move at your weakest position.

Nautilus exercise equipment claims that their machines will provide; (1) both stretching and pre-stretching in the starting position, (2) resistance in the full-contracted position, and (3) available resistance in proportion to your strength in every position throughout a full range of possible movements.

In order to accomplish these objectives, Nautilus equipment provides a resistance that rotates on a common axis with the body-part that is directly moved by the muscle being trained. Since available strength varies throughout the movement, due to anatomical structure, Nautilus also provides a full-range exercise regime that instantly and automatically varies the resistance in accordance with changing strength.

The present time, in order to use this training method, it requires a certain financial investment in order to become a member at a Nautilus training centre. However, these training centres are staffed by knowledgeable personnel, who supervise and instruct trainees on various aspects of weight training such as overload, progressive resistance, etc.

NAUTILUS TRAINING PRINCIPLES

General procedures to be followed on all machines where the regular (positive-negative) form of exercise is performed:

1. On any machine where seat adjustments or body positioning can be varied, make certain that the rotational axis of the cam is directly parallel to the rotational axis (joint) of the body part that is being moved.

2. Position your body in a straight aligned manner. Avoid twisting or shifting your weight during the movement.

3. Never squeeze hand grips tightly, but maintain a loose, comfortable grip (a tight grip elevates blood pressure).

4. Life the resistance (positive work) to the count of two . . . pause . . . lower the resistance (negative work) slowly and smoothly while counting to four.

5. For full-range strength and flexibility (and protection against injury) your range of movement on each machine should be as great as possible.

6. Breathe normally. Try not to hold your breath while straining.

7. Perform each exercise for 8 to 12 repetitions.

   A. Begin with a weight you can comfortably do 8 times.
   B. Stay with that weight until you can perform 12 strict repetitions. On the following workout, increase the weight (approximately 5%) and go back to 8 repetitions.
   C. Ideally, on every workout you should progress in repetitions and/or resistance.

8. Exercise the larger muscle groups first and proceed down to the smaller muscle groups (hips, thighs, back, shoulders, chest, arms, and neck).

9. Your entire workout should take from 20 to 30 minutes.

10. The time lapse between exercise sessions should be at least 48 hours and not more than 96 hours.

NAUTILUS STRENGTH TRAINING PROGRAM

- do all exercises slowly
- 3 second concentric phase while exhaling
- 3 second eccentric phase while inhaling

3 Upper body exercises - 3 sets of 8-10 repetitions

i) Pullover - for your latissimus dorsi muscles
ii) Arm Cross - for your pectoralis muscles
iii) Decline Press - for your pectoralis, triceps & deltoid muscles

3 lower body exercises - 3 sets of 10-12 repetitions

i) Hip and Back Machine - for your lower back muscles & gluteal muscles
ii) Multi-purpose Machine - for your quadriceps
iii) Leg Curl Machine - for your hamstrings

3 sets of 25 sit ups - at any point in your program

Pullover - Adjust seat so axis of rotation is through shoulder
- Hips back, fasten seatbelt
- Use safety bar to get in and out of position
- Push on elbow pads. Don't clench bar with hands.
- Go back as far as you can and then pull bar right in to your abdomen.
- Do all 3 sets on this machine (with a 2-3 minute rest between sets) before moving on.

Arm Cross - Use pad behind back if necessary to get axis of rotation. of overhead cams directly over shoulders.
- Adjust seat so elbows flex at approx. 90^{o}.
- Keep forearms verticle, don't clench hand rests.
- Bring arms right together without sitting up.
- Do one set then immediately do Decline Press.
- Wait 2-3 minutes after Decline Press then repeat both exercises for 2 more sets.

Decline Press - Increase resistance from Arm Cross because this is compound press (ie. you are using more muscles).
- Go as far back as possible without taking your hands off the bar.
- Pause at the end of extension.

Hip and Back Machine - Align hips with axis of rotation of cams.
- Fasten seatbelt.
- Start by extending both legs.
- Keeping one leg extended, bring other leg up high. Stretch.
- Bring leg down and extend then bring other leg up high. Stretch.
- Do 10-12 contractions per leg (ie. 20-24 total).
- You can do all 3 sets on this machine first or 1 set on each lower body machine and repeat 3 times.

Multi-Purpose Machine - Rest belt on hip bones.
- Squat to attach ring.
- Choose step according to leg length.
- Stand on balls of feet.
- Keep back straight, head up.
- Squat to 90° then extend to vertical position.

Leg Curl Machine - Lie on stomach with knee caps just over the edge of the board.
- Grab bar with hands.
- Keep bum down.
- Contract hamstrings to bring bar close to bum.

Remember the Overload Principle.
After three days of being able to reach the top of the range (ie. 10 reps for upper body, 12 reps for lower body) increase resistance.